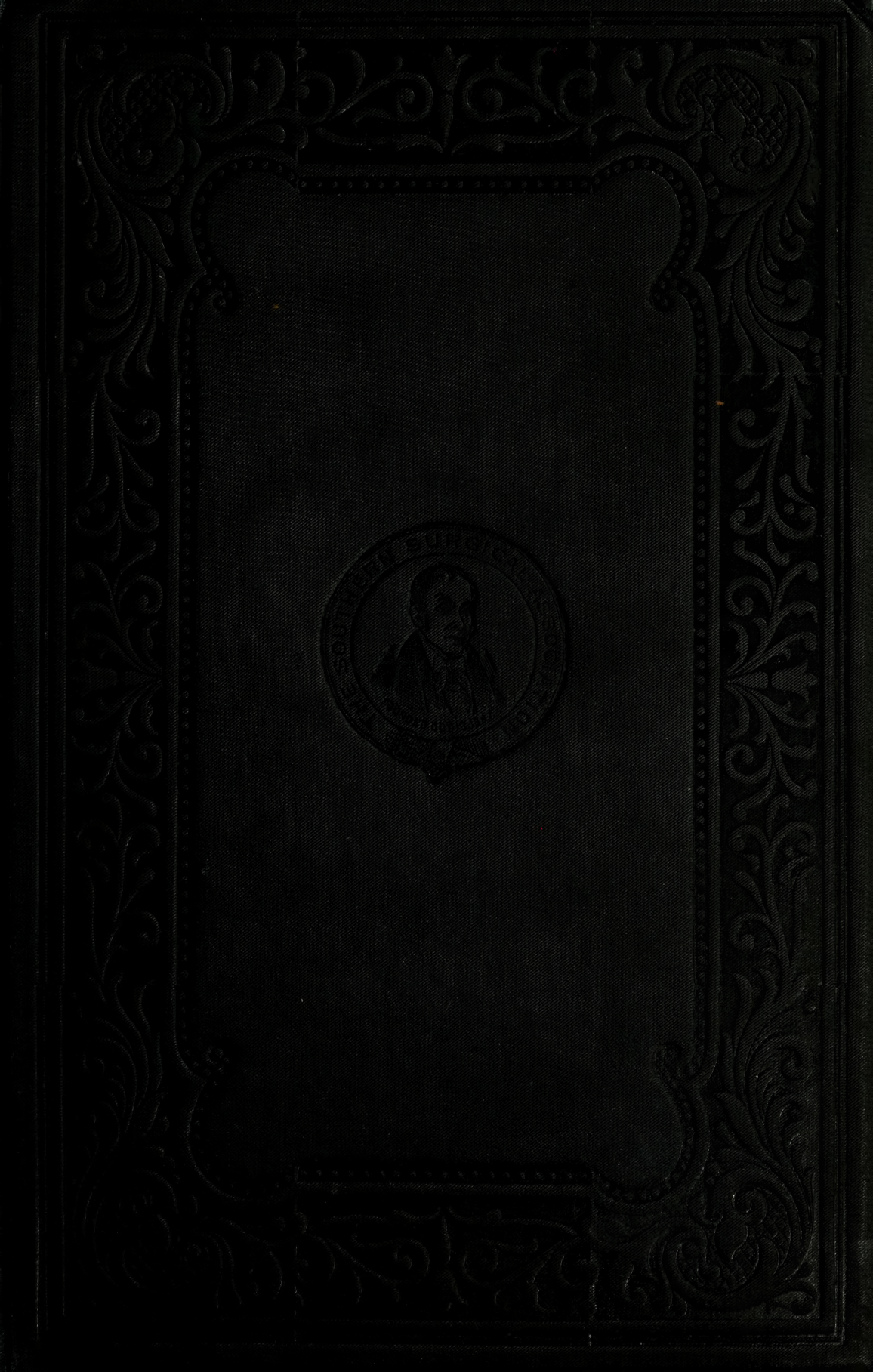

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OF THE

SOUTHERN SURGICAL ASSOCIATION

VOLUME XXX

THIRTIETH SESSION

HELD AT ST. AUGUSTINE, FLORIDA

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PUBLISHED BY THE ASSOCIATION

1918

The Association does not hold itself responsible for the views enunciated in the papers and discussions published in this volume.

H. A. ROYSTER, M.D., *Secretary,*
RALEIGH, N. C.

DORNAN, PRINTER
PHILADELPHIA

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LIST OF OFFICERS

FROM THE ORGANIZATION TO THE PRESENT TIME.

* Deceased.

	President	*Vice-Presidents*	*Secretary*	*Treasurer*
1888.	*W. D. Haggard	R. D. Webb J. W. Sears	*W. E. B. Davis	Hardin P. Cochrane
1889.	*Hunter H. McGuire	W. O. Roberts *Bedford Brown	*W. E. B. Davis	Hardin P. Cochrane
1890.	*George J. Engelmann	*Berthold E. Hadra Duncan Eve	*W. E. B. Davis	Hardin P. Cochrane
1891.	Lewis S. McMurtry	*J. McFadden Gaston J. T. Wilson	*W. E. B. Davis	Hardin P. Cochrane
1892.	*J. McFadden Gaston	*Cornelius Kollock *George Ben Johnston	*W. E. B. Davis	Hardin P. Cochrane
1893.	*Bedford Brown	*Joseph Price George A. Baxter	*W. E. B. Davis	Hardin P. Cochrane
1894.	*Cornelius Kollock	A. B. Miles J. B. S. Holmes	*W. E. B. Davis	Hardin P. Cochrane
1895.	*Louis McLane Tiffany	Ernest S. Lewis *Manning Simons	*W. E. B. Davis	*Richard Douglas
1896.	Ernest S. Lewis	Joseph Taber Johnson *Richard Douglas	*W. E. B. Davis	*A. M. Cartledge
1897.	*George Ben Johnston	W. E. Parker Floyd W. McRae	*W. E. B. Davis	*A. M. Cartledge
1898.	*Richard Douglas	*H. H. Mudd James A. Goggans	*W. E. B. Davis	*A. M. Cartledge
1899.	Joseph Taber Johnson	F. W. Parham *W. L. Robinson	*W. E. B. Davis	*A. M. Cartledge
1900.	*A. M. Cartledge	*Manning Simons W. P. Nicolson	*W. E. B. Davis	W. D. Haggard
1901.	*Manning Simons	George H. Noble L. C. Bosher	W. D. Haggard	Floyd W. McRae
1902.	*W. E. B. Davis	J. Wesley Bovée J. W. Long	W. D. Haggard	Floyd W. McRae
1903.	J. Wesley Bovée	Christopher Tompkins Bacon Saunders	W. D. Haggard	Floyd W. McRae
1904.	Floyd W. McRae	George S. Brown J. Shelton Horsley	W. D. Haggard	Charles M. Rosser
1905.	Lewis C. Bosher	J. D. S. Davis I. S. Stone	W. D. Haggard	Charles M. Rosser
1906.	George H. Noble	Stuart McGuire E. Denegre Martin	W. D. Haggard	Charles M. Rosser
1907.	Howard A. Kelly	Rufus E. Fort Hubert A. Royster	W. D. Haggard	Charles M. Rosser
1908.	F. W. Parham	Henry D. Fry W. F. Westmoreland	W. D. Haggard	Stuart McGuire
1909.	Stuart McGuire	John Young Brown R. S. Cathcart	W. D. Haggard	Wm. S. Goldsmith
1910.	W. O. Roberts	Joseph C. Bloodgood Lewis C. Morris	W. D. Haggard	Wm. S. Goldsmith
1911.	Rudolph Matas	Guy L. Hunner J. Garland Sherrill	W. D. Haggard	Wm. S. Goldsmith
1912.	J. M. T. Finney	J. E. Thompson W. P. Carr	W. D. Haggard	Wm. S. Goldsmith
1913.	John Young Brown	*Ap Morgan Vance Lomax Gwathmey	W. D. Haggard	Le Grand Guerry
1914.	John W. Long	A. C. Scott James F. Mitchell	W. D. Haggard	Le Grand Guerry
1915.	Bacon Saunders	Thomas S. Cullen S. M. D. Clark	W. D. Haggard	Le Grand Guerry
1916.	Thomas S. Cullen	Robert S. Hill Willard Bartlett	W. D. Haggard	Le Grand Guerry
1917.	W. D. Haggard	J. Ernest Stokes F. R. Hagner	H. A. Royster	Le Grand Guerry
1918.	Isaac S. Stone	Le Grand Guerry Carey P. Rogers	H. A. Royster	Guy L. Hunner

LIST OF MEETING PLACES, WITH DATES AND PRESIDING OFFICERS

1888.	Birmingham,	William D. Haggard
1889.	Nashville,	Hunter H. McGuire
1890.	Atlanta,	George J. Engelmann
1891.	Richmond,	Lewis S. McMurtry
1892.	Louisville,	J. McFadden Gaston
1893.	New Orleans,	Bedford Brown
1894.	Charleston,	Cornelius Kollock
1895.	Washington,	Louis McLane Tiffany
1896.	Nashville,	Ernest S. Lewis
1897.	St. Louis,	George Ben Johnston
1898.	Memphis,	Richard Douglas
1899.	New Orleans,	Joseph Taber Johnson
1900.	Atlanta,	A. M. Cartledge
1901.	Richmond,	Manning Simons
1902.	Cincinnati	W. E. B. Davis
1903.	Atlanta,	J. Wesley Bovée
1904.	Birmingham,	Floyd W. McRae
1905.	Louisville,	Lewis C. Bosher
1906.	Baltimore,	George H. Noble
1907.	New Orleans,	Howard A. Kelly
1908.	St. Louis,	F. W. Parham
1909.	Hot Springs,	Stuart McGuire
1910.	Nashville,	William O. Roberts
1911.	Washington,	Rudolph Matas
1912.	Old Point,	John M. T. Finney
1913.	Atlanta,	John Young Brown
1914.	Asheville,	John W. Long
1915.	Cincinnati,	Bacon Saunders
1916.	White Sulphur Springs,	Thomas S. Cullen
1917.	St. Augustine	William D. Haggard

HONORARY FELLOWS

Founder.—Cunningham, Russell M.	Ensley, Ala.
Founder.—Davis, John D. S.	Birmingham, Ala.
1894.—Foy, George	Dublin.
Founder.—Goggans, James A.	Alexander City, Ala.
Founder.—Herff, Ferdinand	San Antonio, Texas.
1894.—Jacobs, Charles	Brussels.
1914.—Johnson, Joseph Taber	Cherrydale, Va.
1910.—Jones, Robert	Liverpool.
1910.—Lane, Arbuthnot	London.
1917.—Lewis, Ernest S.	New Orleans, La.
Founder.—Long, John Wesley	Greensboro N. C.
1917.—McMurtry, Lewis S.	Louisville, Ky.
1894.—Martin, A.	Berlin.
1902.—Maury, Richard B.	Memphis, Tenn.
1894.—Morisani, O.	Naples.
1910.—Morison, Rutherford	New-Castle-on-Tyne.
1910.—Moynihan, B. G. A.	Leeds.
1910.—Myles, Thomas, Sir	Dublin.
1917.—*Polk, William M.	New York, N. Y.
1894.—Pozzi, S.	Paris.
1910.—Robson, Mayo	London.
Founder.—Schilling, Nicholas	Cedar Bayou, Texas.
1910.—Stiles, H. J.	Edinburgh.
Founder.—Stone, Isaac S.	Washington, D. C.
1917.—Tuholske, Herman	St. Louis, Mo.

* Deceased.

FELLOWS

1908.—Abell, Irvin, M.D. Professor of Principles and Practice of Surgery and Clinical Surgery, Medical Department, University of Louisville; Visiting Surgeon, University Hospital, Louisville City Hospital, and St. Mary and Elizabeth Hospital. 1228 Second St., Louisville, Ky.

1911.—Allen, Carroll W., M.D. 1312 Louisiana Ave., New Orleans, La.

1915.—Baer, William Stevenson, F.A.C.S., A.B., M.D. Associate Professor, Orthopedic Surgery, Johns Hopkins University; Associate in Orthopedic Surgery in charge of Orthopedic Department, Johns Hopkins Hospital; Consulting Orthopedic Surgeon, Home for Incurables; Surgeon-in-chief, Children's Hospital; Visiting Orthopedic Surgeon, Union Protestant Infirmary, Hospital for Women of Maryland, and Robert Garrett Hospital for Children; Consulting Orthopedic Surgeon, Cambridge Hospital, Cambridge, Md. 4 West Madison Street, Baltimore, Md.

1917.—Bagley, Charles, Jr., A.B., M.D. Associate in Experimental Neurology Johns Hopkins University. 5 West Chase Street, Baltimore, Md.

1914.—Bailey, Fred. Warren, B.Sc., M.D. Assistant Professor of Surgery, St. Louis University School of Medicine; Associate Chief Surgeon, St. John's Hospital; Visiting Gynecologist, St. Louis City Sanitarium. 633 Metropolitan Building, St. Louis, Mo.

1905.—Baker, James Norment, M.D. Visiting Surgeon, St. Margaret's Hospital; Secretary of the Medical Association of the State of Alabama. 719 Madison Avenue, Montgomery, Alabama.

1916.—Balfour, Donald Church, M.D. Surgeon to St. Mary's Hospital. Rochester, Minn.

1903.—Balloch, Edward A., M.D. Professor of Surgery, Medical Department of Howard University; Surgeon to Freedmen's Hospital; Member American Academy of Medicine. 1511 Rhode Island Avenue, N. W., Washington, D. C.

1916.—Barksdale, John W., M.D. Winona, Miss.

1908.—Barr, Richard Alexander, M.D. Professor of Surgery and Clinical Surgery, Medical Department of Vanderbilt University; late Major of First Tennessee Infantry, U. S. A. First National Bank Building, Nashville, Tenn.

1905.—Barrow, David, M.D. Ex-President Kentucky State Medical Society. 148 Market Street, Lexington, Ky.

1905.—Bartlett, Willard, M.D. Surgeon to St. Anthony's Hospital. *Vice-President,* 1916. 4257 Washington Boulevard, St. Louis, Mo.

1901.—Baughman, Greer, M.D. Professor of Obstetrics, Medical College of Virginia; Visiting Obstetrician to the Virginia Hospital, Memorial Hospital, and Stuart Circle Hospital, Richmond, Virginia. 26 North Laurel Street.

1917.—Bell, Richard Phillip, B.A., M.D. Member of Staff, King's Daughters Hospital. Masonic Building, Staunton, Va.

1907.—Bevan, Arthur Dean, M.D. Professor of Surgery, Rush Medical College, University of Chicago. 100 State Street, Chicago, Ill.

1916.—Biggs, Montgomery Herman, M.D. Surgeon to Rutherford Hospital; Chief Surgeon C. C. and O. Railway. Rutherfordton, N. C.

1916.—Black, William T., M.D. Exchange Building, Memphis, Tenn.

1908.—Blair, Vilray Papin, A.M., M.D. Metropolitan Building, St. Louis, Mo.

1906.—Bloodgood, Joseph Colt, M.D. Associate Professor of Surgery, Johns Hopkins University; Associate Surgeon, Johns Hopkins Hospital; Surgeon to the Union Protestant Infirmary; Chief Surgeon to St. Agnes' Hospital. *Vice-President,* 1910. 904 North Charles Street, Baltimore, Md.

1901.—Boldt, Hermann J., M.D. Gynecologist to Post-Graduate Hospital, St. Mark's Hospital, German Polyclinic; Consulting Gynecologist to the Beth Israel Hospital and to St. Vincent's Hospital; Professor of Gynecology, Post-Graduate Medical School; Member of the Gynecological Society of Germany; Fellow of the American Gynecological Society; Fellow of the Royal Society of Medicine (London); ex-President of the New York Obstetrical Society; of the German Medical Society of New York; of the Section of Obstetrics of the New York Academy of Medicine. 39 East Sixty-first Street, New York, N. Y.

1902.—Bonifield, Charles Lybrand, M.D. Professor of Clinical Gynecology, Medical College of Ohio (Medical Department University of Cincinnati); Member American Association of Obstetricians and Gynecologists; ex-President, Cincinnati Academy of Medicine; ex-President, Cincinnati Obstetrical Society. 409 Broadway, Cincinnati, Ohio.

1890.—Bosher, Lewis Crenshaw, M.D. Professor of Practice of Surgery and Clinical Surgery in the Medical College of Virginia; Visiting Surgeon to the Memorial Hospital; Member of American Surgical Association; Member of the American Association of Obstetricians and Gynecologists; Member of Association of American Anatomists; Member of American Urological Association; ex-President of Richmond Academy of Medicine and Surgery. *Vice-President,* 1901; *President,* 1905. 422 East Franklin Street, Richmond, Va.

1895.—Bovée, J. Wesley, M.D. Fellow of American Gynecological Society; President of Washington Obstetrical and Gynecological Society; ex-President Medical and Surgical Society of the District of Columbia; Honorary Fellow of Medical Society of Virginia; Professor of Gynecology of George Washington University, Washington, D. C.; Gynecologist to Providence, Columbia, and George Washington University Hospitals; Consulting Physician to St. Ann's Infant Asylum, and Attending Gynecologist to St. Elizabeth's Hospital for the Insane, Washington, D. C. *Vice-President,* 1902; *President,* 1903; Member of *Council,* 1903–1906. The Rochambeau, Washington, D. C.

1905.—Brown, John Young, M.D. Formerly Surgeon-in-Charge, St. Louis City Hospital; President of the American Association of Obstetricians and Gynecologists, 1906. *Vice-President,* 1909; *President,* 1913. Metropolitan Building, St. Louis, Mo.

1908.—Bryan, Robert C., M.D. Professor of Genito-urinary Diseases, Medical College of Virginia; Visiting Surgeon to the Virginia Hospital. First and Main Streets, Richmond, Virginia.

1910.—Bryan, W. A., M.D. Professor of Surgery and Clinical Surgery, Vanderbilt University; Professor of Oral Surgery, Dental Department, Vanderbilt University. 146 Eighth Avenue, N., Nashville, Tenn.

1913.—Buist, A. Johnston, M.D. Professor of Gynecology, Medical College of the State of South Carolina; Visiting

Gynecologist, Roper Hospital. 279 Meeting Street, Charleston, S. C.

1907.—Burch, Lucius E., M.D. Professor of Gynecology, Vanderbilt University, Medical Department; Gynecologist, Nashville City Hospital; Captain and Surgeon, N. G. S. T. 706 Church Street, Nashville, Tenn.

1917.—Burnam, Curtis Field, A.B., M.D. Surgeon, Howard A. Kelly Hospital. 1418 Eutaw Place, Baltimore, Md.

1900.—Byford, Henry T., M.D. Professor of Gynecology and Clinical Gynecology, College of Physicians and Surgeons (University of Illinois); Professor of Gynecology, Post-Graduate Medical School of Chicago; Surgeon to Woman's Hospital, etc. Peoples Gas Bldg., Chicago, Ill.

1902.—Caldwell, C. E., A.M., M.D. Clinical Professor of Surgery and Associate Professor of Surgical Anatomy in Medical Department, University of Cincinnati; Surgeon to Cincinnati General Hospital. "The Livingstone," Seventh and Race Streets, Cincinnati, Ohio.

1910.—Caldwell, Robert, M.D. Professor of Surgical Anatomy, Vanderbilt University; ex-President of Nashville Academy of Medicine. 315 Jackson Building, Nashville, Tenn.

1906.—Cannaday, John Egerton, M.D. Former Surgeon-in-Charge to Sheltering Arms Hospital, Hansford, W. Va.; Fellow of the American Association of Obstetricians and Gynecologists; Non-resident Honorary Fellow of the Kentucky State Medical Society; Fellow of the West Virginia Medical Association, Virginia Medical Society, Tri-State Society of Virginia and the Carolinas, American Association of Railway Surgeons. 100 Capitol Street, Charleston, W. Va.

1904.—Carothers, Robert, M.D. Professor of Clinical Surgery, Medical College of Ohio, Medical Department, Cincinnati University; Surgeon to the Good Samaritan Hospital; 413 Broadway, Cincinnati, Ohio.

1903.—Carr, William P., M.D. Professor of Physiology and Professor of Clinical Surgery, Columbian University; Surgeon to the Emergency Hospital and University Hospital; Consulting Surgeon to the Washington Asylum Hospital and the Goverment Hospital for the Insane, Washington, D. C. *Vice-President*, 1912. 1418 L Street, N. W., Washington, D. C.

1899.—Cathcart, R. S., M.D. Professor of Abdominal Surgery, Medical College of the State of South Carolina; Surgeon-in-chief, Roper Hospital, Charleston, S. C.; Surgeon,

Atlantic Coast Line Railway, Charleston Consolidated Railway and Lighting Co., The Citadel—the Military College of South Carolina, and the Confederate Home College, Charleston, S. C. *Vice-President,* 1909. 66 Hasell Street, Charleston, South Carolina.

1907.—Clark, John G., M.D. Professor of Gynecology, University of Pennsylvania; Gynecologist-in-Chief, University Hospital. 2017 Walnut Street, Philadelphia, Penna.

1907.—Clark, S. M. D., M.D. Professor of Gynecology and Clinical Obstetrics, Tulane Medical Department; Visiting Surgeon, Charity Hospital. 1435 Harmony Street, New Orleans, Louisiana.

1893.—Cocram, Henry S., M.D. 108 Baronne Street, New Orleans, La.

1908.—Coffey, Robert C., M.D. Secretary, Oregon State Board of Medical Examiners; Councillor, Oregon State Medical Association. Stevens Building, Portland, Oregon.

1909.—Cole, Herbert Phalon, M.D. Instructor in Gynecology and Clinical Diagnosis, Medical Department of the University of Alabama; Gynecologist to City Hospital of Mobile. 202 Conti Street, Mobile, Ala.

1916.—Coleman, Claude C., M.D. Professor of Oral Surgery and Anesthesia, Associate in Clinical Surgery, Medical College of Virginia; Attending Surgeon to Memorial and Virginia Hospitals. 200 W. Grace Street, Richmond, Va.

1902.—Coley, William B., M.D. Clinical Lecturer on Surgery, College of Physicians and Surgeons; Assistant Surgeon in Hospital for Ruptured and Crippled; Attending Surgeon to General Memorial Hospital; Fellow of the American Surgical Association, New York Surgical Society, and New York Academy of Medicine. 40 East Forty-first Street, New York, N. Y.

1914.—Cousins, William Lewis, M.D. Surgeon-in-Chief, St. Barnabas' Hospital; Consulting Surgeon, Maine General Hospital. 231 Woodford Street, Portland, Maine.

1916.—Cowden, Charles N., M.D. Surgeon to City Hospital; Gynecologist to St. Thomas' Hospital. Hitchcock Bldg., Nashville, Tenn.

1907.—Crawford, Walter W., M.D. Ex-President Southern Medical Association; ex-President Perry County Medical Society; ex-President Mississippi State Medical Association; Surgeon to South Mississippi Infirmary. Hattiesburg, Miss.

1902.—Crile, George W., M.D. Professor of Surgery, The School of Medicine of Western Reserve University; Visiting Surgeon to Lakeside Hospital. 1021 Prospect Avenue, Cleveland, Ohio.

1908.—Crisler, Joseph Augustus, M.D. Professor of Anatomy and Operative Surgery, College of Physcians and Surgeons, Memphis, Tennessee; Lecturer on Minor Surgery, Medical Department, University of Mississippi; ex-Member of Mississippi State Board of Health and Medical Examiners; ex-Vice-President Mississippi State Medical Association and Tri-State Medical Association. Memphis Trust Building, Memphis, Tenn.

1917.—Cullen, Ernest K., M.B. Assistant Professor of Gynecology, Detroit College of Medicine and Surgery; Associate Surgeon in Gynecology, Harper Hospital. David Whitney Building, Detroit, Mich.

1906.—Cullen, Thomas Stephen, M.B. Associate Professor of Gynecology, Johns Hopkins University; Associate in Gynecology, Johns Hopkins Hospital; Consultant in Abdominal Surgery, Church Home and Infirmary. Fellow of the American Gynecological Society; Honorary Member to La Societa Italiana Ostettricia Ginecologia, Rome; Corresponding Member of the Gesellschaft f. Geburtshülfe, Leipzig. *Vice-President*, 1915; *President*, 1916. 20 E. Eager Street, Baltimore, Maryland.

1907.—Danna, Joseph A., M.D. Chief Visiting Surgeon, Charity Hospital. Charity Hospital, New Orleans, Louisiana.

1910.—Davis, John Staige, M.D. Instructor in Surgery, Johns Hopkins University; Assistant Surgeon to the Out-Patient Department of Johns Hopkins Hospital; Visiting Surgeon to the Union Protestant Infirmary, Visiting Surgeon to the Church Home and infirmary. 1200 Cathedral Street, Baltimore, Md.

1900.—Deaver, John B., M.D. Surgeon-in-Chief to the Lankenau Hospital. 1634 Walnut Street, Philadelphia.

1896.—Doughty, W. H., M.D. Professor of Special Surgery and Surgical Pathology, Medical Department, University of Georgia; Chief Surgeon to Charleston and Western Carolina Railroad. 822 Greene Street, Augusta, Ga.

1914.—Downes, William Augustus, M.D. Assistant Professor of Clinical Surgery, Columbia University, College of Physicians and Surgeons; Attending Surgeon, General Memorial

and Babies' Hospitals; Associate Attending Surgeon, St. Luke's Hospital and Hospital for Ruptured and Crippled. 37 West Seventy-first Street, New York, N. Y.

1916.—Du Bose, Francis Goodwin, M.D. Surgeon-in-Charge of Vaughan Memorial Hospital. Selma, Ala.

1890.—Earnest, John G., M.D. Professor of Clinical Gynecology, College of Physicians and Surgeons; formerly Professor of Gynecology, Southern Medical College; Visiting Gynecologist to the Grady Hospital. 828 Candler Building, Atlanta, Ga.

1907.—Elbrecht, Oscar Herman, M.D. Visiting Surgeon, St. Louis City Hospital; Consulting Surgeon to St. Louis Maternity, Bethesda and Missouri Pacific Railroad Hospitals. 423 Metropolitan Building, St. Louis, Mo.

1891.—Elkin, William Simpson, M.D. Professor of Operative and Clinical Surgery, Atlanta College of Physicians and Surgeons; ex-President of the Atlanta Society of Medicine; Surgeon to Grady Hospital. 27 Luckie St., Atlanta, Ga.

1904.—Finney, J. M. T., M.D. Associate Professor of Surgery, Johns Hopkins University; Surgeon to Out-patients, Johns Hopkins Hospital. *President,* 1912. 1300 Eutaw Place, Baltimore, Md.

1917.—Fletcher, Marshall H., M.D. Chief of Staff, Asheville Mission Hospital; Surgeon Clarence Barker Memorial Hospital and Dispensary, Biltmore. 17 Church Street, Asheville, N. C.

1901.—Fort, Rufus E., M.D. Chief Surgeon, Nashville Terminal Company; Surgeon to Tennessee Central Railroad; Surgeon to Nashville Hospital; *Vice-President,* 1907. 209 Seventh Avenue, N., Nashville, Tenn.

1905.—Frank, Louis, M.D. Professor of Surgery and Clinical Surgery, University of Louisville; Visiting Surgeon to Louisville City Hospital. 400 Atherton Building, Louisville, Ky.

1916.—Frazier, Charles H., M.D. Professor of Clinical Surgery, University of Pennsylvania. 1724 Spruce Street, Philadelphia, Pa.

1902.—Freiberg, Albert H., M.D. Professor of Orthopedic Surgery in the Medical College of Ohio, Medical Department of the University of Cincinnati; Orthopedic Surgeon to the Cincinnati Hospital and to the Jewish Hospital of Cincinnati. 19 West Seventh Street, Cincinnati, Ohio.

1917.—Gerster, John C. A., M.D. Instructor in Operative Surgery, Cornell University Medical College. 34 East 75th St., New York, N. Y.

1917.—Gessner, Hermann B., A.M., M.D. Professor of Operative and Clinical Surgery in Tulane University; Chief Visiting Surgeon to Charity Hospital; Junior Surgeon to Touro Infirmary. 1105 Maison Blanche, New Orleans, La.

1913.—Gibbon, R. L., M.D. Charlotte, N. C.

1900.—Goldsmith, W. S., M.D. *Treasurer*, 1909–1912. 262 Jackson Street, Atlanta, Ga.

1892.—Grant, Horace H., M.D. Professor of Surgery in the Hospital College of Medicine; Surgeon to the Louisville City Hospital. 328 Atherton Building, Louisville, Ky.

1905.—Guerry, Le Grand, M.D. Surgeon, Columbia Hospital. *Treasurer*, 1913–1918. *Vice-President*. Columbia, S. C.

1896.—Gwathmey, Lomax, M.D. Attending Gynecologist to St. Vincent's Hospital and Dispensary, *Vice-President*, 1913. Norfolk, Va.

1896.—Haggard, William David, M.D. Professor of Surgery and Clinical Surgery, Vanderbilt University. Surgeon to St. Thomas' Hospital, Vanderbilt and City Hospitals. *Treasurer*, 1900; *Secretary*, 1901–1916; *President*, 1917. 706 Church Street, Nashville, Tenn.

1910.—Hagner, Francis Randall, M.D. Professor of Genito-urinary Surgery, George Washington University; Attending Genito-urinary Surgeon, George Washington University Hospital; Attending Surgeon, Garfield Memorial Hospital. *Vice-President*, 1917. The Farragut, Washington, D. C.

1892.—Hall, Rufus Bartlett, A.M., M.D. Professor of Clinical Gynecology in the Miami Medical College; Gynecologist to the Presbyterian Hospital; ex-President of the Cincinnati Obstetrical Society; ex-President of the American Association of Obstetricians and Gynecologists; Fellow of British Gynecological Society. 628 Elm Street, Cincinnati, O.

1910.—Hanes, Granville S., M.D. Professor of Surgery of the Rectum and Intestinal Diseases, Medical Department of the University of Louisville; Surgeon to the Louisville City Hospital. The Atherton Building, Louisville, Ky.

1896.—Heflin, Wyatt, M.D. Ex-President of the Jefferson County Medical Society. Birmingham, Ala.

1917.—Helms, John Sullivan, A.B., M.D. Chief Surgeon, Tampa Children's Home; Associate Surgeon, Gordon Keller Memorial and Woman's Hospitals. Tampa, Fla.

1905.—Hendon, George A., M.D. Professor of Surgery and Clinical Surgery, University of Louisville. 968 Baxter Avenue, Louisville, Ky.

1889.—Hill, Robert Somerville, M.D. Gynecologist to the Laurel Hill Hospital; Senior Councillor of the Medical Association of the State of Alabama; ex-President of the Montgomery County Medical and Surgical Association; former Surgeon of the Second Regiment, Alabama State Militia. *Vice-President,* 1916. Montgomery, Ala.

1913.—Hodgson, Fred G., M.D. Assistant in Orthopedics, Atlanta Medical College; Orthopedic Surgeon, Wesley Memorial Hospital and MacVicar Hospital of Spelman Seminary; Junior Orthopedic Surgeon, Grady Memorial Hospital. 72 West Peachtree Street, Atlanta, Ga.

1911.—Hogan, Edgar P., M.D. Associate Professor of Gynecology and Abdominal Surgery, Graduate School of Medicine of the University of Alabama; Resident Surgeon and Gynecologist to the Hillman Hospital. 412–14 Empire Building, Birmingham, Ala.

1906.—Holden, Gerry R., M.D. Formerly House Surgeon, Roosevelt Hospital, New York City; formerly Resident Gynecologist, Johns Hopkins Hospital, and Assistant in Gynecology, Johns Hopkins Medical School. 223 West Forsyth Street, Jacksonville, Fla.

1900.—Horsley, John Shelton, M.D. Surgeon-in-Charge of St. Elizabeth's Hospital. *Vice-President,* 1904. 617 West Grace Street, Richmond, Va.

1903.—Hunner, Guy Le Roy, M.D. Associate in Gynecology, Johns Hopkins University; Professor of Genito-urinary Surgery, Woman's Medical College of Baltimore; Gynecologist-in-Chief, St. Agnes' Hospital; Visiting Gynecologist, Church Home and Hebrew Hospital, Baltimore, Frederick City Hospital, Frederick, Md., and Hagerstown Hospital, Hagerstown, Md.; Consulting Gynecologist, Brattleboro Memorial Hospital, Brattleboro, Vt. *Vice-President,* 1911. *Treasurer.* 2305 St. Paul Street, Baltimore, Md.

1900.—Ill, Edward Joseph, M.D. Surgeon to the Woman's Hospital; Medical Director, St. Michael's Hospital; Gynecologist and Supervising Obstetrician, St. Barnabas' Hospital; Consulting Gynecologist to German Hospital and Beth Israel Hospital of Newark, N. J., to All Souls' Hospital, Morristown, N. J., and to Mountain Side Hospital, Montclair, N. J.; Vice-

President from New Jersey to Pan-American Congress of 1893. 1002 Broad Street, Newark, N. J.

1912.—Jackson, William R., M.D. Professor of Surgery to the Medical Department of the University of Alabama; Consulting Surgeon to the City Hospital and Southern Infirmary, Mobile, Ala. 164 St. Michael Street, Mobile, Ala.

1908.—Jonas, Ernst, M.D. Clinical Professor of Surgery, Medical Department, Washington University; Chief of Surgical Clinic, Washington University Hospital; Gynecologist to the Jewish Hospital; Surgeon to the Martha Parsons Hospital for Children; Fellow of the American Association of Obstetricians and Gynecologists. 4495 Westminster Place, St. Louis, Missouri.

1910.—Jones, Edward Groves, A.B., M.D. Professor of Surgery, Medical Department, Emory University (Atlanta Medical College); Surgeon to Grady Memorial Hospital, Georgia Baptist Hospital and Wesley Memorial Hospital. 714 Hurt Building, Atlanta, Ga.

1902.—Jordan, William Mudd, M.D. 501 Woodward Building, Birmingham, Ala.

1912.—Judd, Edward Starr, M.D. Surgeon to St. Mary's Hospital, Rochester, Minn.

1890.—Kelly, Howard Atwood, M.D. Founder of Kensington Hospital, Philadelphia; Associate Professor of Obstetrics, University of Pennsylvania, 1888–89; Professor of Gynecology and Obstetrics, Johns Hopkins University, 1889–99; Professor of Gynecology, Johns Hopkins University; Gynecologist-in-Chief to Johns Hopkins Hospital; Member Associé Etranger de la Société Obstétricale et Gynécologique de Paris; Correspondirendes Mitglied der Gesellschaft f. Geburtshülfe zu Leipzig; Honorary Fellow, Edinburgh Obstetrical Society, Royal Academy of Medicine (Ireland), and British Gynecological Society; Member of Washington Academy of Sciences; Fellow of the American Gynecological Society. *President*, 1907. 1418 Eutaw Place, Baltimore, Maryland.

1917.—Kerr, Harry Hyland, M.D., C.M. Clinical Professor of Surgery, George Washington University Medical School; Attending Surgeon Garfield Memorial and Children's Hospitals; Associate Surgeon, Providence Hospital. 1742 N. St., N. W., Washington, D. C.

1907.—Kirchner, Walter C. G., M.D. Formerly Surgeon-in-Charge, St. Louis City Hospital. 508 Metropolitan Building, St. Louis, Mo.

1899.—Kohlman, William, M.D. 3500 Prytania Street, New Orleans, La.

1913.—LaRoque, G. Paul, M.D. Associate Professor of Surgery, Medical College of Virginia; Surgeon, Memorial Hospital. 501 East Grace Street, Richmond, Va.

1914.—Landry, Lucian, M.D. Assistant Demonstrator in Operative Surgery and Instructor in Clinical Surgery, Tulane University of Louisiana, School of Medicine; Junior Surgeon, Touro Infirmary; Visiting Surgeon, Charity Hospital. 2255 St. Charles Avenue, New Orleans, La.

1916.—Law, Arthur Ayer, M.D. Associate Professor of Surgery, University of Minnesota Medical School and Mayo Foundation; Major, M. O. R. C. and Director, University of Minnesota Base Hospital, No. 26; Surgeon, University of Minnesota Hospital and University Hospital; Assistant Chief of University Hospital. Syndicate Building, Minneapolis, Minn.

1917.—Ledbetter, Samuel L., Jr., M.D. First National Bank Building, Birmingham, Ala.

1894.—Leigh, Southgate, M.D. Visiting Surgeon to St. Vincent's and Norfolk Protestant Hospitals; First Vice-President of Medical Society of Virginia, 1899-1900. 147 Granby Street, Norfolk.

1912.—Lewis, Bransford, M.D. Century Building, St. Louis, Mo.

1914.—Lewis, Dean, A.B., M.D. Associate Professor of Surgery, Rush Medical College; Attending Surgeon, Presbyterian Hospital; Consulting Surgeon, Anne W. Durand Hospital of the Memorial Institute for Infectious Diseases. 122 South Michigan Avenue, Chicago, Ill.

1900.—Lloyd, Samuel, M.D. Professor of Surgery, New York Post-Graduate Medical School; Attending Surgeon to the Post-Graduate Hospital and St. Francis' Hospital, and Consulting Surgeon to the Benedictine Sanitarium at Kingston, N. Y. 12 West Fiftieth Street, New York, N. Y.

1904.—Lupton, Frank A., M.D. 816 Woodward Building, Birmingham, Ala.

1902.—McAdory, Wellington P., M.D. Surgeon to Hillman Hospital. Birmingham, Ala.

1895.—McGannon, M. C., M.D. Professor of Surgery and Clinical Surgery, Medical Department, Vanderbilt University; Chief Surgeon, Woman's Hospital. 118 Eighth Avenue, N., Nashville, Tenn.

1911.—McGlannan, Alexius, M.D. Associate Professor of Surgery, College of Physicians and Surgeons, Baltimore. 114 West Franklin St., Baltimore, Md.

1893.—McGuire, Stuart, M.D. Professor of the Principles of Surgery and Clinical Surgery, University College of Medicine; Surgeon-in-Charge, St. Luke's Hospital; Visiting Surgeon, Virginia Hospital; Consulting Surgeon, Virginia Home for Incurables. *Vice-President*, 1906; *Treasurer*, 1908; *President*, 1909. 518 East Grace Street, Richmond, Va.

1891.—McGuire, W. Edward, M.D. Professor of Gynecology, University College of Medicine; Gynecologist to the Virginia Hospital. 411 East Grace Street, Richmond, Va.

1888.—McRae, Floyd Wilcox, M.D. Professor of Gastro-intestinal, Rectal, and Clinical Surgery, Atlanta College of Physicians and Surgeons; Surgeon to Grady Hospital; ex-President Medical Association of Georgia; ex-Secretary of Section on Surgery and Anatomy, American Medical Association. *Vice-President*, 1897; *Treasurer*, 1901–1903; *President*, 1904. Peters Building, Atlanta, Ga.

1905.—MacLean, Henry Stuart, M.D. Professor of Pathology, University College of Medicine. 406 West Grace Street, Richmond, Va.

1915.—Maes, Urban, M.D. Assistant Professor of Clinical and Operative Surgery, Tulane University of Louisiana School of Medicine; Junior Surgeon, Charity Hospital and Touro Infirmary; Senior Surgeon, New Orleans Dispensary for Women and Children. 1671 Octavia Street, New Orleans, Louisiana.

1912.—Malone, Battle, M.D. 1701 Exchange Building, Memphis, Tenn.

1891.—Marcy, Henry Orlando, M.D., LL.D. Ex-President of the American Medical Association; President of the Section of Gynecology, Ninth International Medical Congress, late President of the American Academy of Medicine; Corresponding Member of the Medico-Chirurgical Society of Bologna, Italy; Fellow of the American Association of Obstetricians and Gynecologists. 180 Commonwealth Avenue, Boston, Mass.

1899.—Martin, E. Denegre, M.D. Professor of Surgery, Post-Graduate Department, Tulane Medical College; Visiting Surgeon to Charity Hospital. *Vice-President*, 1906. 741 Carondelet St., New Orleans, La.

1909.—Martin, Frank, M.D. Clinical Professor of Surgery at the University of Maryland. 1000 Cathedral Street, Baltimore, Md.

1913.—Martin, Franklin H., M.D. Professor of Gynecology, Post-Graduate School; Consulting Staff, St. Luke's Hospital. Editor, *Surgery, Gynecology and Obstetrics*. 30 North Michigan Avenue, Chicago, Ill.

1904.—Mason, J. M., M.D. Gynecologist to St. Vincent's Hospital. 1915 Sixteenth Avenue, S., Birmingham, Ala.

1899.—Mastin, William M., M.D. Fellow of the American Surgical Association; Member of the American Association of Genito-urinary Surgeons; Surgeon to the Mobile City Hospital. Joachim and Conti Streets, Mobile, Ala.

1893.—Matas, R., M.D. Professor of Surgery, Medical Department of Tulane University; Fellow of the American Surgical Association. *President*, 1911. 3523 Prytania St., New Orleans, La.

1896.—Mathews, William P., M.D. Professor of Anatomy, Medical College of Virginia; Orthopedic Surgeon to the Old Dominion Hospital. Richmond, Va.

1917.—Maury, John M., M.D. Professor of Gynecology, University of Tennessee College of Medicine; Gynecologist, Memphis General and Baptist Memorial Hospitals. Bank of Commerce Building, Memphis, Tenn.

1902.—Mayo, Charles H., M.D. Attending Surgeon, St. Mary's Hospital; Fellow of American Surgical Society; ex-President, Western Surgical and Gynecological Association and of Minnesota State Medical Society. Rochester, Minn.

1888.—Miller, C. Jeff., M.D. Visiting Surgeon to Charity Hospital of Louisiana; Professor of Clinical Obstetrics and Clinical Gynecology in the Tulane University of Louisiana; Chief of the Division of Obstetrics, Gynecology and Abdominal Surgery of the Presbyterian Hospital. 1638 Joseph Street, New Orleans, La.

1916.—Miller, Edward Howe, Jr., M.D. Surgeon, Danville General Hospital; Assistant Surgeon, Southern Railway. 6 Chestnut Place, Danville, Va.

1917.—Miller, Robert Talbott, Jr., M.D. Professor of Surgery, University of Pittsburgh Medical School; Surgeon to Mercy Hospital. Diamond Bank Building, Pittsburgh, Pa.

1907.—Mitchell, James F., M.D. Surgeon to Providence Hospital. *Vice-President*, 1914. 1344 Nineteenth Street, Washington, D. C.

1901.—Mixter, Samuel Jason, M.D. Surgeon, Massachusetts General Hospital; Consulting Surgeon, Massachusetts Charity Eye and Ear Infirmary; Instructor of Surgery, Harvard Medical School. 180 Marlborough St., Boston, Mass.

1910.—Moore, James E., M.D. Professor and Chief of the Department of Surgery in the College of Medicine and Surgery of the University of Minnesota; Fellow and ex-Vice-President of the American Surgical Association; Member and ex-President of the Western Surgical Association; Member and ex-Chairman of the Surgical Section of the American Medical Association; Honorary Member of the American Orthopedic Association. University of Minnesota, Minneapolis, Minn.

1911.—Moore, John Thomas, M.D. Kress Medical Bldg., Houston, Texas.

1904.—Moran, John F., M.D. Professor of Obstetrics, Georgetown University; Obstetrician to Georgetown University Hospital and Columbia Hospital for Women. 2426 Pennsylvania Avenue, Washington, D. C.

1902.—Morris, Lewis C., M.D. Professor of Anatomy and Associate Professor of Gynecology, Birmingham Medical College; Councillor of Medical Association, State of Alabama. *Vice-President,* 1910. 1203–6 Empire Building, Birmingham, Ala.

1900.—Morris, Robert Tuttle, M.D. Professor of Surgery, New York Post-Graduate Medical School and Hospital. 616 Madison Avenue, New York, N. Y.

1901.—Mullally, Lane, M.D. Professor of Obstetrics and Diseases of Children, Medical College, State of South Carolina; Visiting Physician in Obstetrics and Pediatrics, Roper Hospital. Charleston, S. C.

1916.—Murphy, Fred Towsley, M.D. Professor of Surgery, Washington University Medical School; Surgeon-in-Chief to the Barnes and St. Louis Children's Hospitals; Consulting Surgeon to the St. Louis City Hospital. Euclid Avenue and Kingshighway, St. Louis, Mo.

1916.—Newell, E. Dunbar, M.D. Surgeon to the Newell and Newell Sanitarium; Consulting Surgeon to the Erlanger Hospital. Chattanooga, Tenn.

1889.—Nicolson, William Perrin, M.D. Professor of Anatomy and Clinical Surgery, Atlanta College of Physicians and Surgeons; Visiting Surgeon to Grady Hospital; Senior Surgeon to Wesley Memorial Hospital; Senior Surgeon, St. Joseph's Hospital. *Vice-President,* 1900. Healey Building, Atlanta, Ga.

1890.—Noble, George H., M.D. Dean and Professor of Abdominal Surgery and Clinical Gynecology, Atlanta School of Medicine; Gynecologist of Grady Hospital; Senior Gynecologist, Wesleyan Memorial Hospital, Atlanta; Fellow of the American Gynecological Association and the American Association of Obstetricians and Gynecologists; ex-President of the Medical Association of Georgia; ex-Secretary of the Section of Obstetrics, American Medical Association. *Vice-President*, 1901; *President*, 1906. 131 South Pryor Street, Atlanta, Ga.

1911.—Norris, Henry, M.D. Rutherfordton, N. C.

1901.—Ochsner, A. J., M.D. Adjunct Professor of Clinical Surgery, College of Physicians and Surgeons; Surgeon-in-Chief, Augustana and St. Mary's Hospital. 2106 Sedgwick Street, Chicago, Ill.

1903.—Ochsner, Edward H., M.D. Surgeon to Augustana and St. Mary's Hospital. 2106 Sedgwick Street, Chicago, Illinois.

1902.—Oliver, John Chadwick, M.D. Dean and Professor of Operative Surgery, Miami Medical College; Surgeon to the Cincinnati, Christ, and Presbyterian Hospitals. 628 Elm Street, Cincinnati, Ohio.

1888.—Parham, F. W., M.D. Professor of Surgery in the New Orleans Polyclinic. *Vice-President*, 1899; *President*, 1908. 1429 Seventh Street, New Orleans, La.

1910.—Payne, Robert Lee, Jr., M.D. Assistant Gynecologist to St. Vincent's Hospital; Surgeon to the Southern Railway; Consulting Surgeon to the Seaboard Air Line Railway. 300 Freemason St., Norfolk, Va.

1916.—Peck, Charles H., M.D. Professor of Clinical Surgery, Columbia University; Surgeon to Roosevelt Hospital. 30 W. Fiftieth Street, New York, N. Y.

1899.—Perkins, W. M., M.D. Chief of the Clinic Chair of General Clinical and Operative Surgery, New Orleans Polyclinic; Assistant Demonstrator of Operative Surgery, Medical Department, Tulane University of Louisiana; Visiting Surgeon to the Charity Hospital. 830 Canal Street, New Orleans, Louisiana.

1908.—Peterson, Reuben, A.M., M.D. Professor of Obstetrics and Gynecology, University of Michigan; Obstetrician and Gynecologist-in-Chief to the University of Michigan Hospital. Ann Arbor, Mich.

1917.—Pool, Eugene H., M.D. Clinical Professor of Surgery, Columbia University, College of Physicians and Surgeons; Attending Surgeon, New York Hospital. 107 East Sixtieth Street, New York, N. Y.

1900.—Porter, Miles F., M.A., M.D. Surgeon to Hope Hospital; Professor of Surgery, Indiana University School of Medicine. 47 West Wayne Street, Fort Wayne, Ind.

1916.—Pryor, Stewart Wylie, M.D. Chief Surgeon to the Pryor Hospital; Chief Surgeon to C. and N. W. and L. and C. Railways; Division Surgeon to S. A. L. Railway. Chester, S. C.

1900.—Ransohoff, Joseph, M.D., F.R.C.S. Professor of Anatomy and Clinical Surgery, University of Cincinnati; Surgeon to Cincinnati, Good Samaritan, and Jewish Hospitals; Member of American Surgical Association. 19 West Seventh Street, Cincinnati, Ohio.

1911.—Reder, Francis, M.D. Surgeon, St. Louis City and St. John's Hospitals and Missouri Sanatorium. Delmar Building, St. Louis, Mo.

1890.—Reed, Charles Alfred Lee, M.D. Ex-President of the American Medical Association; Professor of Gynecology and Abdominal Surgery in the Cincinnati College of Medicine and Surgery; Surgeon to the Cincinnati Free Hospital for Women; Fellow of the American Association of Obstetricians and Gynecologists; Fellow of the British Gynecological Society; ex-Chairman of the Section on Obstetrics and Diseases of Women of the American Medical Association; Secretary-General of the Pan-American Medical Congress, 1893; Honorary Member of the Medical Society of the State of New York. 19 W. Seventh Street, Cincinnati, Ohio.

1914.—Rhodes, Goodrich Barbour, A.B., M.D. Assistant Clinical Professor of Surgery, Ohio-Miami Medical College of the University of Cincinnati; Junior Attending Surgeon, Cincinnati and Good Samaritan Hospitals and Episcopal Hospital for Children. 4 West Seventh Street, Cincinnati, Ohio.

1917.—Rhodes, Robert L., A.B., M.D. 1103–5 Lamar Building, Augusta, Ga.

1912.—Richardson, Edward P., M.D. Surgeon to Outpatients, Massachusetts General Hospital, and Surgeon to the Robert B. Brigham Hospital for Chronic Diseases. 224 Beacon Street, Boston, Mass.

1888.—Roberts, William O., M.D. Professor of Principles of Surgery and Clinical Surgery, Medical Department of the

University of Louisville; formerly Secretary of the Surgical Section of the American Medical Association. *Vice-President,* 1889; *President,* 1910. Atherton Building, Louisville, Ky.

1901.—Robins, Charles Russell, M.D. Professor of Gynecology, Medical College of Virginia; Gynecologist to the Memorial Hospital; Surgeon to the Atlantic Coast Railroad. 8 West Grace Street, Richmond, Va.

1917.—Robinson, Julian McG., M.D. 753 Main Street, Danville, Va.

1906.—Rogers, Carey Pegram, M.D. Chief of Surgical Staff, St. Luke's Hospital; Consulting Surgeon, Seaboard Air Line Railway. *Vice-President.* 221 Laura Street, Jacksonville, Florida.

1901.—Rogers, Mack, M.D. Professor of Anatomy, Birmingham Medical College. 212 Twentieth Street, Birmingham, Alabama.

1901.—Rosser, Charles M., M.D. Professor of Surgery, Baylor University, College of Medicine; Consulting Surgeon, Parland Hospital; Attending Surgeon, Texas Baptist Memorial Sanitarium; ex-Vice-President Texas State Medical Association; Vice-President, Tri-State Society of Texas, Louisiana, and Arkansas. *Treasurer,* 1904–07. 432 Gaston Avenue, Dallas, Texas.

1899.—Royster, Hubert Ashley, A.B., M.D. Surgeon to Rex Hospital; Surgeon-in-Chief, St. Agnes' Hospital; Surgeon to Southern Railway. *Vice-President,* 1907. *Secretary.* 423 Fayetteville Street, Raleigh, N. C.

1909.—Ruffin, Kirkland, M.D. Surgeon-in-Charge of St. Christopher's Hospital. 218 York Street, Norfolk, Va.

1908.—Russell, Wm. Wood, M.D. Associate Professor of Gynecology, Johns Hopkins University; Associate in Gynecology, Johns Hopkins Hospital; Gynecologist to Union Protestant Infirmary, Baltimore. 1208 Eutaw Place, Baltimore, Md.

1893.—Saunders, Bacon, M.D. Professor of Surgery and Clinical Surgery and Dean of Faculty of Medical Department, Fort Worth University; ex-President, Texas State Medical Association. *Vice-President,* 1903; *Member of Council,* 1908; *President,* 1915. Flatiron Bldg., Fort Worth, Texas.

1907.—Scott, Arthur Carroll, M.D. Senior Surgeon, Temple Sanatorium. *Vice-President,* 1914. Temple, Texas.

1915.—Seelig, Major G., A.B., M.D. Professor of Surgery, St. Louis University School of Medicine; Attending Surgeon,

Jewish and St. Louis City Hospitals; Chief of Surgical Out-Patient Department, Jewish Hospital. Wall Building, St. Louis, Mo.

1899.—Shands, A. R., M.D. Professor of Orthopedic Surgery in the Medical Department of the George Washington University and in the University of Vermont; Orthopedic Surgeon to the George Washington University Hospital, the Emergency Hospital, and Central Dispensary; Charter Member, Washington Academy of Sciences; Member of American Orthopedic Association; and Honorary Member of Medical Society of Virginia. 901 Sixteenth Street, N. W., Washington, D. C.

1914.—Shands, Harley R., A.B., M.D. Visiting Surgeon, Mississippi State Charity and Baptist Hospitals. Century Building, Jackson, Miss.

1917.—Sherk, Henry H., M.D. Central Building, Pasadena, Cal.

1902.—Sherrill, J. Garland, M.D. Professor of Surgery and Clinical Surgery, University of Louisville; Consulting Surgeon to Louisville Hospital. *Vice-President,* 1911. Atherton Building, Louisville, Ky.

1900.—Simpson, Frank Farrow, M.D. Assistant Gynecologist, Mercy Hospital; Gynecologist to Out-patient Department, Mercy Hospital. Jenkins Arcade Bldg., Pittsburgh, Pa.

1913.—Simpson, James Knox, M.D. Attending Gynecologist, St. Luke's Hospital; Associate Gynecologist, Duval County Hospital, Jacksonville, Fla.

1917.—Sistrunk, W. E., M.D. Mayo Clinic, Rochester, Minnesota.

1899.—Smythe, Frank David, M.D. Gynecologist to St. Joseph's and City Hospitals; Professor of Gynecology, Didactic and Clinical, in the Memphis Hospital Medical College; ex-President West Tennessee Medical and Surgical Association; formerly Member of the Mississippi State Board of Health and State Board of Medical Examiners of Mississippi. Porter Building, Memphis, Tenn.

1916.—Souchon, Marion, M.D. House Surgeon, Hotel Dieu; Surgeon French Hospital; Assistant Professor of Clinical Surgery, Tulane University; Visiting Surgeon, Charity Hospital. Whitney Bank Bldg., New Orleans, La.

1904.—Stokes, James Ernest, M.D. *Vice-President,* 1917. Salisbury, N. C.

1894.—Talley, Dyer F., M.D. Associate Professor of Surgery, Birmingham Medical College; Attending Surgeon to Hillman Hospital; Surgeon to the Talley and McAdory Infirmary; ex-President of the Jefferson County Medical Society; Member of the Southern Medical Association; Member of the Pan-American Medical Association; Member of the Alumni Association of the Charity Hospital of Louisiana; Member of the Board of Censors of the Jefferson County Medical Society; Member of the Alabama State Board of Censors, Committee of Public Health and Examiners; Fellow of the American Association of Obstetricians and Gynecologists. 1801 Seventh Avenue, Birmingham, Ala.

1892. Thompson, J. E., M.D. Professor of Surgery in the University of Texas. *Vice-President*, 1912. Galveston, Tex.

1916.—Thorning, W. Burton, M.D. Chief Surgeon of the Gulf Coast Lines. Kress Building, Houston, Texas.

1908.—Torrance, Gaston, M.D. Member Surgical Staff Hillman Hospital; also the Sisters' Hospital and St. Vincent's; Fellow of the American Association of Obstetricians and Gynecologists. 328 Woodward Building, Birmingham, Ala.

1909.—Trout, Hugh Henry, M.D. Surgeon-in-Chief of Jefferson Surgical Hospital, Roanoke, Va. 1303 Franklin Road, Roanoke, Va.

1917.—Tupper, Paul Y., M.D. Clinical Professor of Surgery, Washington University Medical School; Visiting Surgeon St. Luke's Hospital; Consulting Surgeon, Missouri Baptist Sanitarium, Bethesda, Jewish and St. John's Hospitals, St. Louis Eye, Ear, Nose and Throat Infirmary. Wall Building, St. Louis, Mo.

1912.—Turck, Raymond C., M.D. Consolidated Building, Jacksonville, Fla.

1893.—Vander Veer, Albert, M.D. Professor of Clinical, Didactic, and Abdominal Surgery, Albany Medical College; ex-President of the American Surgical Association; ex-President of the American Association of Obstetricians and Gynecologists; ex-President of the Medical Society of the State of New York. 28 Eagle Street, Albany, New York.

1909.—Vaughan, George Tully, M.D. Professor of Surgery and Head of Department of Surgery in Georgetown University; Chief Surgeon in Georgetown University Hospital; Visiting Surgeon, Tuberculosis Hospital; Consulting Surgeon, St. Elizabeth's Hospital; Consulting Surgeon, Washington Asylum Hospital. 1718 I Street, Washington, D. C.

1912.—Walker, George, M.D. Associate in Surgery, Johns Hopkins University. 1 E. Centre St., Baltimore, Maryland.

1911.—Waring, T. P., M.D. Savannah, Ga.

1905.—Wathen, John R., A.B., M.D. Professor of Principles and Practice of Surgery and Clinical Surgery, University of Louisville; Surgeon to St. Anthony's, Louisville City, and University of Louisville Hospitals. 628 Fourth Avenue, Louisville, Ky.

1898.—Watkins, Isaac Lafayette, M.D. Ex-President of the Montgomery County Medical and Surgical Society. Montgomery, Ala.

1907.—Watts, Stephen H., M.D. Professor of Surgery University of Virginia; Surgeon-in-Chief and Director of the University of Virginia Hospital. University of Virginia, Charlottesville, Va.

1888.—Westmoreland, Willis F., M.D. Professor of Surgery, Atlanta College of Physicians and Surgeons. *Vice-President*, 1908. 241 Equitable Building, Atlanta, Ga.

1907.—Willis, A. Murat, M.D. Instructor in Abdominal Surgery, Medical College of Virginia; Junior Surgeon to Memorial Hospital, Richmond, Va. 405 East Grace Street, Richmond, Va.

1913.—Windell, James Tolbert, M.D. 715 West Jefferson Street, Louisville, Ky.

1905.—Winslow, Randolph, M.D., LL.D. Professor of Surgery, University of Maryland; Chief Surgeon, University Hospital; Consulting Surgeon, Hebrew Hospital, and to Hospital for Crippled Children. 1900 Mt. Royal Terrace, Baltimore, Md.

1905.—Witherspoon, T. Casey, M.D. Formerly Professor of Operative and Clinical Surgery, Medical Department, St. Louis University; Member of American Association of Anatomists. 307 West Granite Street, Butte, Mont.

1900.—Wysor, John C., M.D. Surgeon-in-Charge, Chesapeake and Ohio Hospital. Clifton Forge, Va.

1900.—Young, Hugh H., M.D. Clinical Professor of Urology, Johns Hopkins University Medical Department. 330 North Charles Street, Baltimore, Md.

1802.—Zinke, E. Gustav, M.D., F.A.C.S. Professor of Obstetrics and Clinical Midwifery in the Ohio-Miami Medical College, University of Cincinnati, 1896–1916; Emeritus Pro-

fessor of Obstetrics, 1916; Honorary Chief of Staff and Obstetrician and Gynecologist to the German Hospital; President of the Cincinnati Obstetric Society, 1887; President, Academy of Medicine of Cincinnati, 1894; Member and Chairman of Section on Obstetics, Gynecology and Abdominal Surgery, American Medical Association, 1914; Fellow (and Secretary) of American Association of Obstetricians and Gynecologists; Honorary Member, Jackson County Medical Society, Kansas City, Mo.; Honorary Member, Cincinnati Obstetricians and Gynecologists, 1908. 4 West Seventh Street, Cincinnati, Ohio.

GEOGRAPHICAL LIST OF FELLOWS

ALABAMA

Baker, James Norment, 719 Madison Ave., Montgomery.
Cole, Herbert Phalon, 202 Conti St., Mobile.
Du Bose, Francis Goodwin, Selma.
Heflin, Wyatt, Birmingham.
Hill, Robert Somerville, Montgomery.
Hogan, Edgar P., 412–14 Empire Bldg., Birmingham.
Jackson, William R., 164 St. Michael St., Mobile.
Jordan, William Mudd, 501 Woodward, Bldg., Birmingham.
Ledbetter, Samuel L., Jr., First National Bank Bldg., Birmingham.
Lupton, Frank A., 816 Woodward Bldg., Birmingham.
McAdory, Wellington P., Birmingham.
Mason, J. M., 1915 Sixteenth Ave., S., Birmingham.
Mastin, William M., Joachim and Conti Sts., Mobile.
Morris, Lewis C., 1203–6 Empire Bldg., Birmingham.
Rogers, Mack, 212 Twentieth St., Birmingham.
Talley, Dyer F., 1801 Seventh Ave., Birmingham.
Torrance, Gaston, 328 Woodward Bldg., Birmingham.
Watkins, Isaac LaFayette, Montgomery.

CALIFORNIA

Sherk, Henry H., Central Building, Pasadena.

FLORIDA

Helms, John Sullivan, Tampa.
Holden, Gerry R., 223 W. Forsyth St., Jacksonville.
Rogers, Carey Pegram, 221 Laura St., Jacksonville.
Simpson, James Knox, Jacksonville.
Turck, Raymond C., Consolidated Bldg., Jacksonville.

GEORGIA

Doughty, W. H., 822 Greene St., Augusta.
Earnest, John G., 828 Candler Bldg., Atlanta.

Elkin, William Simpson, 27 Luckie St., Atlanta.
Goldsmith, W. S., 262 Jackson St., Atlanta.
Hodgson, Fred. G., 728 Candler Bldg., Atlanta.
Jones, Edward Groves, 714 Hurt Bldg., Atlanta.
McRae, Floyd Wilcox, Peters Bldg., Atlanta.
Nicholson, William Perrin, 1900 Healy Bldg., Atlanta.
Noble, George H., 131 S. Pryor St., Atlanta.
Rhodes, Robert L., 1103–5 Lamar Bldg., Augusta.
Waring, T. P., Savannah.
Westmoreland, Willis F., 241 Equitable Bldg., Atlanta.

ILLINOIS

Bevan, Arthur Dean, 100 State St., Chicago.
Byford, Henry T., Peoples Gas Bldg., Chicago.
Lewis, Dean D., 122 S. Michigan Ave., Chicago.
Martin, Franklin H., 30 North Michigan Ave., Chicago.
Ochsner, A. J., 2106 Sedgwick Street, Chicago.
Ochsner, Edward H., 2106 Sedgwick St., Chicago.

INDIANA

Porter, Miles F., 47 W. Wayne St., Fort Wayne.

KENTUCKY

Abell, Irvin, 1228 Second St., Louisville.
Barrow, David, 148 Market St., Lexington.
Frank, Louis, 400 Atherton Bldg., Louisville.
Grant, Horace H., 329 Atherton Bldg., Louisville.
Hanes, Granville S., Atherton Bldg., Louisville.
Hendon, George A., 968 Baxter Ave., Louisville.
Roberts, William O., Atherton Bldg., Louisville.
Sherrill, J. Garland, Atherton Bldg., Louisville.
Wathen, John R., 628 Fourth Ave., Louisville.
Windell, James T., 715 West Jefferson St., Louisville.

LOUISIANA

Allen, Carroll W., 1312 Louisiana Ave., New Orleans.
Clark, S. M. D., 1435 Harmony St., New Orleans.
Cocram, Henry S., 108 Baronne St., New Orleans.
Danna, Joseph A., Charity Hospital, New Orleans.

Gessner, Herman B., 1105 Maison Blanche Bldg., New Orleans.
Kohlman, William, 3500 Prytania St., New Orleans.
Landry, Lucian, 2255 St. Charles Ave., New Orleans.
Maes, Urban, 1671 Octavia St., New Orleans.
Martin, E. Denegre, 741 Carondelet St., New Orleans.
Matas, Rudolph, 3523 Prytania St., New Orleans.
Miller, C. Jeff, 1638 Joseph St., New Orleans.
Parham, F. W., 1429 Seventh St., New Orleans.
Perkins, W. M., 830 Canal St., New Orleans.
Souchon, Marion, Whitney Bank Bldg., New Orleans.

MAINE

Cousins, William Lewis, 231 Woodford St., Portland.

MARYLAND

Baer, William S., 4 W. Madison St., Baltimore.
Bagley, Charles, Jr., 5 W. Chase St., Baltimore.
Bloodgood, Joseph C., 904 N. Charles St., Baltimore.
Burnam, Curtis Field, 1418 Eutaw Place, Baltimore.
Cullen, Thomas Stephen, 20 E. Eager St., Baltimore.
Davis, John Staige, 1200 Cathedral St., Baltimore.
Finney, J. M. T., 1300 Eutaw Place, Baltimore.
Hunner, Guy LeRoy, 2305 St. Paul St., Baltimore.
Kelly, Howard Atwood, 1418 Eutaw Place, Baltimore.
McGlannan, Alexius, 114 W. Franklin St., Baltimore.
Martin, Frank, 1000 Cathedral St., Baltimore.
Russell, William Wood, 1208 Eutaw Place, Baltimore.
Walker, George, 1 E. Centre St., Baltimore.
Winslow, Randolph, 1900 Mount Royal Terrace, Baltimore.
Young, Hugh H., 330 N. Charles St., Baltimore.

MASSACHUSETTS

Marcy, Henry Orlando, 180 Commonwealth Ave., Boston.
Mixter, Samuel Jason, 180 Marlborough St., Boston.
Richardson, Edward P., 224 Beacon St., Boston.

MICHIGAN

Cullen, Ernest K., David Whitney Bldg., Detroit.
Peterson, Reuben, Ann Arbor.

MINNESOTA

Balfour, Donald Church, Rochester.
Judd, Edward Starr, Rochester.
Law, Arthur Ayer, Syndicate Bldg., Minneapolis.
Mayo, Charles H., Rochester.
Moore, James E., University of Minnesota, Minneapolis.
Sistrunk, W. E., Rochester.

MISSISSIPPI

Barksdale, John W., Winona.
Crawford, Walter W., Hattiesburg.
Shands, Harley R., Century Bldg., Jackson.

MISSOURI

Bailey, Fred W., 633 Metropolitan Bldg., St. Louis.
Bartlett, Willard, 4257 Washington Blvd., St. Louis.
Blair, Vilray Papin, Metropolitan Bldg., St. Louis.
Brown, John Young, Metropolitan Bldg., St. Louis.
Elbrecht, O. H., 423 Metropolitan Bldg., St. Louis.
Jonas, Ernst, 4495 Westminster Place, St. Louis.
Kirchner, Walter C. G., 508 Metropolitan Bldg., St. Louis.
Lewis, Bransford, Century Bldg., St. Louis.
Murphy, Fred. Towsley, Euclid Ave. and Kingshighway, St. Louis.
Reder, Francis, Delmar Bldg., St. Louis.
Seelig, Major G., Wall Bldg., St. Louis.
Tupper, Paul Y., Wall Bldg., St. Louis.

MONTANA

Witherspoon, T. Casey, 307 West Granite Street, Butte.

NEW JERSEY

Ill, Edward Joseph, 1002 Broad St., Newark.

NEW YORK

Boldt, Herman J., 39 E. 61st St., New York City.
Coley, William B., 40 E. 41st St., New York City.

Downes, William Augustus, 37 W. 71st St., New York City.
Gerster, John C. A., 34 E. 75th St., New York City.
Lloyd, Samuel, 12 E. 50th St., New York City.
Morris, Robert Tuttle, 616 Madison Ave., New York City.
Peck, Charles H., 30 W. 50th St., New York City.
Pool, Eugene H., 107 E. 60th St., New York City.
Vander Veer, Albert, 28 Eagle St., Albany.

NORTH CAROLINA

Biggs, Montgomery Herman, Rutherfordton.
Fletcher, Marshall H., 17 Church St., Asheville.
Gibbon, R. L., Charlotte.
Norris, Henry, Rutherfordton.
Royster, Hubert Ashley, 423 Fayetteville St., Raleigh.
Stokes, James Ernest, Salisbury.

OHIO

Bonifield, Charles Lybrand, 409 Broadway, Cincinnati.
Caldwell, C. E., The Livingstone, 7th and Race Sts., Cincinnati.
Carothers, Robert, 413 Broadway, Cincinnati.
Crile, George W., 1021 Prospect Ave., Cleveland.
Freiberg, Albert H., 19 W. Seventh St., Cincinnati.
Hall, Rufus Bartlett, 628 Elm St., Cincinnati.
Oliver, John Chadwick, 628 Elm St., Cincinnati.
Ransohoff, Joseph, 19 W. 7th St., Cincinnati.
Reed, Charles Alfred Lee, 19 W. 7th St., Cincinnati.
Rhodes, Goodrich Barbour, 4 W. Seventh St., Cincinnati.
Zinke, E. Gustav, 4 W. Seventh St., Cincinnati.

OREGON

Coffey, Robert C., Stevens Building, Portland.

PENNSYLVANIA

Clark, John G., 2017 Walnut St., Philadelphia.
Deaver, John B., 1634 Walnut St., Philadelphia.
Frazier, Charles H., 1724 Spruce St., Philadelphia.
Miller, Robert Talbott, Jr., Diamond Bank Bldg., Pittsburgh.
Simpson, Frank Farrow, Jenkins Arcade Bldg., Pittsburgh.

SOUTH CAROLINA

Buist, A. Johnston, 279 Meeting Street, Charleston.
Cathcart, R. S., 66 Hasell St., Charleston.
Guerry, Le Grand, Columbia.
Mullally, Lane, Charleston.
Pryor, Stewart Wylie, Chester.

TENNESSEE

Barr, Richard Alexander, First National Bank Bldg., Nashville.
Black, William T., Exchange Bldg., Memphis.
Bryan, W. A., 146 Eighth Ave., N., Nashville.
Burch, Lucius E., 706 Church St., Nashville.
Caldwell, Robert, 315 Jackson Bldg., Nashville.
Cowden, Charles N., Hitchcock Bldg., Nashville.
Crisler, Joseph Augustus, Memphis Trust Bldg., Memphis.
Fort, Rufus E., 209 Seventh Ave., N., Nashville.
Haggard, William David, 706 Church St., Nashville.
McGannon, M. C., 118 Eighth Ave., N., Nashville.
Malone, Battle, 1701 Exchange Bldg., Memphis.
Maury, John M., Bank of Commerce Bldg., Memphis.
Newell, E. Dunbar, Chattanooga.
Smythe, Frank David, Porter Bldg., Memphis.

TEXAS

Moore, John Thomas, Kress Bldg., Houston.
Rosser, Charles M., 432 Gaston Ave., Dallas.
Saunders, Bacon, Flatiron Bldg., Fort Worth.
Scott, Arthur Carroll, Temple.
Thompson, J. E., Galveston.
Thorning, W. Burton, Kress Bldg., Houston.

VIRGINIA

Baughman, Greer, 26 N. Laurel St., Richmond.
Bell, Richard Phillip, Masonic Bldg., Staunton.
Bosher, Lewis Crenshaw, 422 E. Franklin St., Richmond.
Bryan, Robert C., First and Main Sts., Richmond.
Coleman, Claude C., 200 W. Grace St., Richmond.
Gwathmey, Lomax, Norfolk.
Horsley, John Shelton, 617 W. Grace St., Richmond.

La Roque, G. Paul, 501 East Grace St., Richmond.
Leigh, Southgate, 147 Granby St., Norfolk.
McGuire, Stuart, 518 E. Grace St., Richmond.
McGuire, W. Edward, 411 E. Grace St., Richmond.
MacLean, Henry Stuart, 406 W. Grace St., Richmond.
Mathews, William P., Richmond.
Miller, Edward Howe, Jr., 6 Chestnut Place, Danville.
Payne, Robert Lee, Jr., 300 Freemason St., Norfolk.
Robins, Charles Russell, 8 W. Grace St., Richmond.
Robinson, Julian McG., 753 Main St., Danville.
Ruffin, Kirkland, 218 York St., Norfolk.
Trout, Hugh Henry, 1303 Franklin Road, Roanoke.
Watts, Stephen H., Charlottesville.
Willis, A. Murat, 405 E. Grace St., Richmond.
Wysor, John C., Clifton Forge.

WEST VIRGINIA

Cannaday, John Egerton, 100 Capitol St., Charleston.

WASHINGTON, D. C.

Balloch, Edward A., 1511 Rhode Island Ave., N. W.
Bovée, J. Wesley, The Rochambeau.
Carr, William P., 1418 L. Street, N. W.
Hagner, Francis Randal, The Farragut.
Kerr, Harry Hyland, 1742 N Street, N. W.
Mitchell, James F., 1344 Nineteenth St.
Moran, John F., 2426 Pennsylvania Ave.
Shands, A. R., 901 Sixteenth Street, N. W.
Vaughan, George Tully, 1718 I St.

CONSTITUTION

ARTICLE I

The name of this Association shall be THE SOUTHERN SURGICAL ASSOCIATION.

ARTICLE II

The object of this Association shall be to further the study and practice of Surgery in its various departments, especially among the profession of the Southern States.

ARTICLE III

SECTION 1. The qualifications which must be possessed by an applicant for fellowship in the Association shall be: (*a*) Good standing in his State Association; (*b*) experience covering not less than ten years of professional work; (*c*) practice limited essentially to surgery or one of its specialties; (*d*) recommendation by three Fellows, two preferably from his own State.

SECTION 2. Application for Fellowship shall be filed with the Secretary on the blank form provided for that purpose. The Council at each annual session shall carefully consider all candidates and may nominate one for each vacancy that exists on the roll for election or rejection by the Association.

ARTICLE IV

The Honorary Fellows of the Association shall not exceed twenty-five in number. They shall be nominated by the Council and elected by the Association. They will not be required to pay any dues and shall enjoy all the privileges of other Fellows except that of voting or holding office; this exception does not apply to Founders.

ARTICLE V

SECTION 1. The officers of this Association shall be a President, two Vice-Presidents, a Secretary, a Treasurer, and a Council, elected by ballot.

SEC. 2. The President and Vice-Presidents shall be elected for one year, and the President shall not be eligible for reëlection at any time; the Secretary and Treasurer, each, for five years; and the Council as provided for in the By-laws.

ARTICLE VI

SECTION 1. It shall be the duty of the President to preside at all meetings of the Association; to give the casting vote; to see that the rules of order and decorum be properly enforced in all deliberations of the Association; to sign the approved proceedings of each meeting, and to approve such orders as may be drawn upon the Treasurer for expenditures ordered by the Association.

SEC. 2. In the absence of the President the first Vice-President shall preside, and in his absence the second Vice-President shall preside.

SEC. 3. In the absence of all three, the Association shall elect one of its Fellows to preside *pro tem.*

SEC. 4. It shall be the duty of the Secretary to keep a true and correct record of the proceedings of the meetings; to preserve all books, papers, and articles belonging to the archives of the Association; to attest all orders drawn on the Treasurer for moneys appropriated by the Association; to keep the account of the Association with its Fellows; to keep a register of the Fellows, with the dates of their admission and places of residence. He shall collect all moneys due from the Fellows and pay to the Treasurer, taking his receipt for the same. He shall report such unfinished business of previous meetings as may appear on his books requiring action, and attend to such other business as the Association may direct. He shall also supervise and conduct all the correspondence of the Association, and edit the TRANSACTIONS under the direction of the Council.

SEC. 5. It shall be the duty of the Treasurer to keep a correct record of all moneys received from the hands of the Secretary, giving his receipt for the same; pay them out by order of the Association as indorsed by the President and attested by the seal in the hands of the Secretary.

Sec. 6. It shall be the duty of the President of the Association to appoint an Auditing Committee, consisting of three Fellows of the Association, whose duty it shall be to examine the books of the Secretary and Treasurer, and report on the same on the last day of the session.

ARTICLE VII

Vacancies occurring in the offices of the Association shall be filled by appointment of the President until the next meeting. He shall also have the appointment of all committees not otherwise provided for.

ARTICLE VIII

This Constitution shall take effect immediately from the time of its adoption, and shall not be amended except by a written resolution, which shall lie over one year, and receive a vote of two-thirds of the members present.

ARTICLE IX

The Fellowship of the Association shall be limited to two hundred.

BY-LAWS

ARTICLE I

The Southern Surgical Association shall meet annually on the Tuesday of the week preceding the week in which Christmas occurs, at 10 A.M., at such place as may be designated at the preceding meeting.

ARTICLE II

The Fellows present shall constitute a quorum for business.

ARTICLE III

The annual dues of each Fellow shall be $15, paid in advance.

ARTICLE IV

The usual parliamentary rules governing deliberative bodies shall govern the business workings of this Association.

ARTICLE V

All questions before the Association shall be determined by a majority of the votes present.

ARTICLE VI

The President shall deliver an annual address at each meeting of the Association.

ARTICLE VII

The Secretary of the Association shall receive at each annual Session a draft from the President, drawn on the Treasurer, for such sum as may be voted him by the Council, for services rendered the Association, and to this shall be added the necessary expense incurred in the discharge of his official duties.

ARTICLE VIII

It shall be the duty of the Secretary, one month prior to the annual meeting, to notify the Fellows of the Association and urge their attendance.

ARTICLE IX

The authors of papers shall notify the Secretary, six weeks prior to the meeting, of the titles of their essays, so that they may be incorporated in the preliminary programme.

ARTICLE X

COUNCIL

The Council shall consist of five members; and of those elected at the primary meeting, the first shall serve five years, the second four, and the third three, the fourth two, and the fifth one year; so that subsequently one member of the Council shall be elected annually to serve five years. No member of the Council shall be eligible for reëlection. The President and Secretary shall be *ex-officio* members of the Council.

This Council shall organize by electing a Chairman and Secretary, and shall keep a record of its proceedings.

The duties of this Council shall be—

1. To investigate applications for membership and report to the Association the names of such persons as are deemed worthy.

2. To take cognizance of all questions of an ethical, judicial, or personal nature, and upon these the decision of the Council shall be final; *provided*, that appeal may be taken from such decision of the Council to the Association, under a written protest, which protest shall be sustained by the Association, and the matter shall then be referred to a special committee, with power to take final action.

3. All motions and resolutions before the Association shall be referred to the Council without debate, and it shall report by recommendation at as early an hour as possible.

ARTICLE XI

The President shall appoint at each annual meeting a Committee of Arrangements.

ARTICLE XII

The Council shall have full power to omit from the published TRANSACTIONS, in part or in whole, any paper that may be referred to it by the Association, unless specially instructed to the contrary by the Association, which will be determined by vote.

ARTICLE XIII

The Council shall have power to drop from the roll of Fellows any member who fails to pay his dues for more than one year, or misses three consecutive sessions of the Association, or who for any reason fails to retain his good standing in his State association.

ARTICLE XIV

No paper shall be read before this Association which does not deal strictly with a subject of surgical or gynecological importance.

ARTICLE XV

No paper read before this Association shall be published in any medical journal or pamphlet for circulation, as having been read before the Association, without having received the endorsement of the Council.

ARTICLE XVI

The readings of papers shall be limited to twenty minutes each, except by permission of the Association.

MINUTES OF THE PROCEEDINGS

AT THE

THIRTIETH ANNUAL MEETING

OF THE

SOUTHERN SURGICAL ASSOCIATION

HELD AT THE

ALCAZAR HOTEL

St. Augustine, Florida

DECEMBER 18, 19 AND 20, 1917

THIRTIETH ANNUAL MEETING

FIRST DAY—*Tuesday, December,* 18, 1917

The following named members were present:

ABELL, IRVIN.....Louisville, Ky.
BAILEY, FRED WARREN.....St. Louis, Mo.
BLACK, WILLIAM T.....Memphis, Tenn.
BOSHER, LEWIS CRENSHAW.....Richmond, Va.
CALDWELL, ROBERT.....Nashville, Tenn.
CANNADAY, JOHN EGERTON.....Charleston, W. Va.
CLARK, JOHN G.....Philadelphia, Pa.
COLE, HERBERT PHALON.....Mobile, Ala.
COLEMAN, CLAUDE C.....Richmond, Va.
COWDEN, CHARLES N.....Nashville, Tenn.
CULLEN, THOMAS S.....Baltimore, Md.
DANNA, JOSEPH A.....New Orleans, La.
DOUGHTY, W. H.....Augusta, Ga.
DU BOSE, FRANCIS GOODWIN....Selma, Ala.
GUERRY, LE GRAND.....Columbia, S. C.
HAGGARD, WILLIAM D.....Nashville, Tenn.
HELFIN, WYATT.....Birmingham, Ala.
HENDON, GEORGE A.....Louisville, Ky.
HILL, ROBERT SOMERVILLE.....Montgomery, Ala.
HOGAN, EDGAR P.....Birmingham, Ala.
HOLDEN, GERRY R.....Jacksonville, Fla.
HORSLEY, JOHN SHELTON.....Richmond, Va.
HUNNER, GUY LEROY.....Baltimore, Md.
JACKSON, WILLIAM R.....Mobile, Ala.
JORDAN, WILLIAM MUDD.....Birmingham, Ala.
LEWIS, BRANSFORD.....St. Louis, Mo.
LONG, JOHN WESLEY (HON.).....Greensboro, N. C.
LUPTON, FRANK A.....Birmingham, Ala.
MCGLANNAN, ALEXIUS.....Baltimore, Md.
MCGUIRE, STUART.....Richmond, Va.
MARTIN, FRANKLIN H.....Chicago, Ill.

Mason, J. M. . . . . Birmingham, Ala.
Miller, C. Jeff . . . . . New Orleans, La.
Miller, Edward H., Jr. . . . . Danville, Va.
Moore, James E. . . . . Minneapolis, Minn.
Moore, John T. . . . . Houston, Texas.
Morris, Lewis C. . . . . Birmingham, Ala.
Newell, E. Dunbar . . . . . Chattanooga, Tenn.
Ochsner, Edward H. . . . . Chicago, Ill.
Parham, F. W. . . . . New Orleans, La.
Payne, Robert Lee, Jr. . . . . Norfolk, Va.
Ransohoff, Joseph . . . . . Cincinnati, Ohio.
Reder, Francis . . . . . St. Louis, Mo.
Robins, Charles Russell. . . . . Richmond, Va.
Rogers, Carey Pegram . . . . . Jacksonville, Fla.
Royster, Hubert Ashley . . . . . Raleigh, N. C.
Saunders, Bacon . . . . . Forth Worth, Texas.
Shands, A. R. . . . . Washington, D. C.
Simpson, James K. . . . . Jacksonville, Fla.
Stokes, James Ernest . . . . . Salisbury, N. C.
Stone, Isaac S. (Hon.) . . . . . Washington, D. C.
Talley, Dyer F. . . . . Birmingham, Ala.
Thompson, J. E. . . . . Galveston, Texas.
Thorning, W. Burton . . . . . Houston, Texas.
Trout, Hugh Henry . . . . . Roanoke, Va.
Turck, Raymond C. . . . . Jacksonville, Fla.
Watts, Stephen H. . . . . Charlottesville, Va.
Willis, A. Murat . . . . . Richmond, Va.
Windell, James Tolbert . . . . . Louisville, Ky.
Winslow, Randolph. . . . . Baltimore, Md.
Zinke, E. Gustav . . . . . Cincinnati, Ohio.

Letters and messages of regret were received from the following fellows who were unable to attend the meeting: Joseph C. Bloodgood, Baltimore, Maryland; Charles H. Mayo, Rochester, Minnesota; Lucius E. Burch, Nashville, Tennessee; Granville S. Hanes, Louisville, Kentucky; Charles M. Rosser, Dallas, Texas; John D. S. Davis, Birmingham, Alabama; John Staige Davis, Baltimore, Maryland; W. P. Carr, Washington, D. C.; Edward J. Ill, Newark, New Jersey; William Kohlmann, New Orleans, Louisiana; Ernst Jonas, St. Louis, Missouri; John W. Barksdale, Winona, Mississippi; Robert C. Bryan, Richmond, Virginia; Robert T. Morris, New York City; William B. Coley,

New York City; Albert Vander Veer, Albany, New York; Frank D. Smythe, Memphis, Tennessee; J. Wesley Bovée, Washington, D. C.; Henry O. Marcy, Boston, Massachusetts; Lewis S. McMurtry, Louisville, Kentucky; S. M. D. Clark, New Orleans, Louisiana; Herman Tuholske, St. Louis, Missouri, and David Barrow, Lexington, Kentucky.

Morning Session. The Association met in the Assembly Hall of the Alcazar Hotel and was called to order at 10 A.M. by the Secretary, Dr. Hubert A. Royster. He presented Dr. Carey P. Rogers, Jacksonville, Florida, Chairman of the Committee of Arrangements, who made the following announcements of social events:

Tuesday, December 18, at 2.15 P.M., automobile drive about St. Augustine, showing points of interest; reception at the home of Dr. Andrew Anderson, 4.30 to 5.30 P.M., immediately following the drive; tea for the ladies the next afternoon and the annual Association dinner Wednesday evening. Complimentary golf privileges were extended to the Fellows and their guests.

After the announcements, the Secretary presented the President, Dr. William D. Haggard, Nashville, Tennessee, who took official charge of the meeting and stated that the Association was ready to proceed with the reading of papers and discussion thereon.

The following gentlemen were admitted to the courtesies of the floor: Dr. J. M. Robinson, Danville, Virginia; Dr. M. W. Seagars, St. Augustine, Florida; Dr. E. S. Estes, Dr. Iva S. Youmans and Dr. French, Jacksonville, Florida.

Papers were read as follows:

1. "The Value of Biopathological Standardization" (by invitation), by Dr. William C. McCarty, Rochester, Minnesota, which was discussed by Drs. Cullen, Horsley, and in closing by the essayist.

2. "The Development and Perfection of the Interposition Operation for the Relief of Prolapse of the Uterus and Bladder," illustrated by lantern slides, by Dr. I. S. Stone, Washington, D. C.

This paper was discussed by Drs. Martin, Robins, Cullen, Reder, Hunner, and discussion closed by the essayist.

3. "The Causes and Surgical Aspect of Rupture of the Uterus During Labor," by Dr. E. Gustav Zinke, Cincinnati, Ohio.

4. "Cesarean Section under Local Anesthesia," by Dr. Hugh H. Trout, Roanoke, Virginia.

These two papers were discussed together by Drs. Stone, Parham, Long, Mason, Cole, Robins, after which the discussion was closed by the authors of the papers.

5. "Intra-abdominal Hernia," by Dr. F. W. Bailey, St. Louis, Missouri.

On motion, the Association adjourned until 8 P.M.

NOTE.—In the afternoon the members and guests took an automobile drive about St. Augustine, taking in points of scenic and historic interest.

Evening Session. The Association reassembled at 8 P.M., and was called to order by the President.

6. "The Treatment of Certain Forms of Subacute Pancreatitis," by Stephen H. Watts, Charlottesville, Virginia.

Discussed by Drs. DuBose, McGlannan, Morris, Ransohoff, and Cannaday.

7. "Traumatic Rupture of the Spleen with Report of Four Cases," by Dr. A. Murat Willis, Richmond, Virginia.

Discussed by Drs. Ochsner, Du Bose, and discussion closed by the essayist.

8. "Reconstruction of the Choledochus," by Dr. Le Grand Guerry, Columbia, South Carolina.

Discussed by Dr. Horsley, and discussion closed by the essayist.

9. "Long Resections of the Intestine," by Dr. J. E. Cannaday, Charleston, West Virginia.

Discussed by Drs. Ransohoff and Martin.

10. "One Hundred Cases of Inoperable Cancer of the Uterus Treated by Radium," by Dr. John G. Clark, Philadelphia, Pennsylvania.

Discussed by Drs. C. Jeff. Miller, Hunner, Ransohoff, Horsley, Simpson, and in closing by the essayist.

On motion, the Association adjourned until 9 A.M. Wednesday.

SECOND DAY.—*Wednesday, December,* 19, 1917

Morning Session. The Association met at 9.20 A.M., and was called to order by the President.

11. "The Etiology of Ureteral Calculus," by Dr. Guy Le Roy Hunner, Baltimore, Maryland.

12. "The Recurrence of Stone in the Kidney," by Dr. Charles R. Robins, Richmond, Virginia.

These two papers were discussed together by Drs. Lewis,

Thompson, Payne, Jackson, Ochsner, and Ransohoff, after which the discussion was closed by the essayists.

13. "Subparietal Injuries of the Kidney," by Dr. J. M. Mason, Birmingham, Alabama.

Discussed by Drs. Watts, McGlannan, Hunner, Guerry, Royster, Du Bose and Jackson, after which the discussion was closed by the essayist.

The President appointed as Auditing Committee Drs. George A. Hendon, R. L. Payne, Jr., and Robert Caldwell.

In the absence of Dr. J. M. T. Finney and Dr. John Young Brown, members of the Council, the President appointed Drs. F. W. Parham and Stuart McGuire to fill these vacancies pro tem.

14. "The Cancer Problem," by Dr. Charles H. Mayo, Rochester, Minnesota, was read by the Secretary in the absence of the author.

15. "Some Recent Literature Concerning Shock and Its Management," by Dr. F. W. Parham, New Orleans, Louisiana.

Discussed by Drs. Horsley, McCarty, Jordan, Cole, Du Bose, and discussion closed by the essayist.

16. "Retrograde Peristalsis in the Second and Third Portions of the Dilated Duodenum," by Dr. W. Burton Thorning, Houston, Texas.

On motion, the Association adjourned until 2 P.M.

Afternoon Session. The Association reassembled at 2 P.M., and was called to order by the President.

17. "Fracture of the Long Bones; A Clinical Study," by Dr. Francis Reder, St. Louis, Missouri.

18. "Arthrodesis and Bone Graft, with Illustrative Cases," by Dr. F. G. Du Bose, Selma, Alabama.

19. "A Brief Description of the Technic of Making Celluloid Splints," by Dr. A. R. Shands, Washington, D. C.

These three papers were discussed jointly by Drs. Cannaday, McGlannan and Reder, after which the discussion was closed by Dr. Du Bose.

20. "Severe Head Injuries," by Dr. R. L. Payne, Jr., Norfolk, Virginia.

Discussed by Drs. McGlannan, Jackson, Reder, Thompson, and in closing by the essayist.

21. "Consideration of Injuries of the Cervical Spine," by Dr. Joseph Ransohoff, Cincinnati, Ohio.

Discussed by Drs. Moore, Thompson, Jackson, Hendon, and in closing by the essayist.

Adjourned.

Evening Session. After the annual dinner, the Association was called to order by Vice-president J. Ernest Stokes, Salisbury, North Carolina.

President William D. Haggard was introduced and delivered an address on "The Medical Profession and the Great War."

This address was listened to with marked attention and evoked round after round of applause throughout its delivery.

Dr. Franklin H. Martin, Chicago, Illinois, Member of the Council of National Defense, spoke on "The Activities of the Medical Section of the Council of National Defense."

Dr. Edward Martin, Philadelphia (by invitation), spoke on "Some of the Newer Antiseptics," his remarks being accompanied by slides and motion pictures.

On motion, the Association adjourned until Thursday, 9 A.M.

Third Day.—*Thursday, December* 20, 1917

Morning Session. The Association met at 9.15 A.M., and was called to order by the President.

Dr. George A. Hendon reported for the Auditing Committee, stating that the Committee had carefully examined the accounts of the Secretary and the Treasurer and had found them correct in every detail and the books well kept.

It was moved and seconded that the report of the Auditing Committee be adopted. Carried.

The Secretary presented the report of the Council, as follows:

Report of the Council

The Council met December 19, 1917, in the Hotel Alcazar with the following members present: Drs. Saunders, Long, Cullen, Parham (pro tem.), McGuire (pro tem.), the President and Secretary, ex-officio.

I. The Council advises that changes in the Constitution as proposed last year by the special committee, and which were automatically laid over for a year, be ratified for adoption by the Association.

These changes are as follows:

1. Change the title from the Southern Surgical and Gynecological Association to the Southern Surgical Association and

change word Member to Fellow and Membership to Fellowship wherever they occur in the Constitution or By-Laws.

2. Substitute the following for Article II of the present Constitution: The object of this Association shall be to further the study and practice of surgery in its various departments, especially among the profession of the Southern States.

Substitute the following for Article III and Article IV of the present Constitution:

3. Article III, Section 1. The qualifications which must be possessed by an applicant for Fellowship in the Association shall be:

1. Good standing in his State association.

2. Experience covering not less than ten years of professional work.

3. Practice limited essentially to surgery or one of its specialties.

4. Recommendation by three Fellows, two preferably from his own State.

Section 2. Application for Fellowship shall be filed with the Secretary on the blank form provided for that purpose. The Council at each annual session shall carefully consider all candidates and may nominate one for each vacancy that exists on the roll for election or rejection by the Association.

Article IV. The Honorary Fellows of the Association shall not exceed twenty-five in number. They shall be nominated by the Council and elected by the Association. They will not be required to pay any dues and shall enjoy all the privileges of other Fellows, except that of voting or holding office; this exception does not apply to Founders.

II. It is recommended that the names of Drs. William M. Polk, New York City; Lewis S. McMurtry, Louisville, Kentucky; E. S. Lewis, New Orleans, and Herman Tuholske, St. Louis, Missouri, be transferred to the honorary list.

III. The following Fellows are nominated for membership on the Board of Governors of the American College of Surgeons: Dr. Joseph Ransohoff, Cincinnati, Ohio; Dr. Charles R. Robins, Richmond, Virginia; Dr. W. Burton Thorning, Houston, Texas.

IV. According to the rules of the Association in regard to non-attendance seven Fellows are dropped from the roster, as follows: Dr. Walter Brewster Platt, Baltimore, Maryland; Dr. W. C. Borden, Washington, D. C.; Dr. Charles M. Hazen, Richmond, Virginia; Dr. Espy M. Williams, Patterson, Louisi-

ana; Dr. John F. Oechsner, New Orleans, Louisiana; Dr. W. W. Taylor, Memphis, Tennessee and Dr. T. P. Whaley, Charleston, South Carolina.

It is recommended that the resignations of the following Fellows be accepted: Dr. J. M. Hundley, Baltimore, Maryland; Dr. P. E. Michinard, New Orleans, Louisiana; Dr. Edwin Walker, Evansville, Indiana, and Dr. J. Whitridge Williams, Baltimore, Maryland.

V. The following are nominated for membership:

Dr. E. K. Cullen, Detroit, Mich.
Dr. Charles Bagley, Baltimore, Md.
Dr. C. F. Burnam, Baltimore, Md.
Dr. J. A. C. Gerster, New York City.
Dr. R. P. Bell, Staunton, Va.
Dr. S. L. Ledbetter, Birmingham, Ala.
Dr. J. S. Helms, Tampa, Fla.
Dr. W. E. Sistrunk, Rochester, Minn.
Dr. H. H. Sherk, Pasadena, Cal.
Dr. E. H. Pool, New York City.
Dr. P. Y. Tupper, St. Louis, Mo.
Dr. J. M. Maury, Memphis, Tenn.
Dr. J. M. Robinson, Danville, Va.
Dr. Herman B. Gessner, New Orleans, La.
Dr. R. T. Miller, Pittsburgh, Pa.
Dr. G. B. Rhodes, Augusta, Ga.
Dr. H. H. Kerr, Washington, D. C.
Dr. M. H. Fletcher, Asheville, North Carolina.

VI. It is ordered that the deaths of our Fellows, L. McLane Tiffany, George Ben Johnston and F. E. Baldridge be recorded after our usual custom.

VII. It is suggested that Baltimore be the next place of meeting, and that Dr. Thomas S. Cullen be the Chairman of the Committee of Arrangements.

VIII. The following officers are nominated: President, Dr. Isaac S. Stone, Washington, D. C.; Vice-presidents, Dr. Le Grand Guerry, Columbia, South Carolina and Dr. Carey P. Rogers, Jacksonville, Florida; Secretary, Dr. Hubert A. Royster, Raleigh, North Carolina; Treasurer, Dr. Guy Le Roy Hunner, Baltimore, Maryland.

The name of Dr. William D. Haggard is recommended as a member of the Council to succeed Dr. J. M. T. Finney, whose term expires at this session.

IX. The Council recommends that the President and the Secretary shall have the power to appoint an assistant Secretary or Treasurer if either incumbent is called to military service.

Respectfully submitted,

F. W. Parham,
Stuart McGuire,
J. W. Long,
Bacon Saunders,
Thomas S. Cullen.

The President: You have heard the recommendations contained in the report of the Council. What is your pleasure?

Dr. Robert S. Hill: I move that the report be accepted. Seconded.

Dr. John Wesley Long: Before you put the motion, Mr. President, there is one point in the deliberations of the Council which possibly has been overlooked, on account of our having so many changes to make, and I would like to call attention to that point before a vote is taken. It relates to the transfer of the older men to the list of Honorary Fellows. According to the provisions in the By-Laws, they are not permitted to either vote or hold office. While it is true that does not apply specifically to the Founders, yet, at the same time, I would feel loath to put the name of such a man as our distinguished Fellow, Dr. McMurtry (and I use him as an illustration) in such a position that he would not be allowed under the Constitution or the By-Laws to vote or even to hold office. So, it seems to me, sir, it would not be wise to have that apply to men of the class whom we are now placing upon the roll of Honorary Fellows.

The President: To overcome that difficulty the By-Law could be supplemented by adding the words: "This exception does not apply to Founders or to those who have been active as members in the last two years."

Dr. Long: I desire to make such a motion. Seconded.

The President: Such a change in the By-Law would have to be recommended by the Council first, and then brought before the Association. Under the circumstances, I imagine that we cannot have another meeting of the Council.

Dr. Long: Amendments to the Constitution and By-Laws have to come through the Council. This, however, is simply an amendment to the report of the Council as presented, and can be acted on now.

The President: The Chair would be glad to construe your motion as applying to the present report.

The amendment was accepted, and the original motion as amended was put and carried.

The President appointed Drs. Long and Parham to escort the newly elected President, Dr. I. S. Stone, to the platform.

President Haggard, in introducing his successor, said: It affords me very peculiar pleasure to present to you a man who is one of the few to survive of the original founders of this great Association. It seems extremely fitting here on the thirtieth anniversary of the birth of the Association that we would be able to deliver it back into the hands of one of its deliverers at its birth. It is known to you, Dr. Stone, but perhaps it is not known to some of the members, particularly those who have come in since the foundation of this Association, that the gavel of our Association which we treasure very much indeed was made from a leg of the operating table of J. Marion Sims and presented to us. Dr. Stone has in a peculiar way carried out the traditions of the specialty and ideals of that great master of surgery, and I take great pleasure and peculiar pride in transferring this gavel to the hands of one under whom we feel the Association will be carried to a greater destiny than ever before.

Dr. Stone, in accepting the Presidency, said: Mr. President: You know how readily my heart comes up and throbs when anything is said in the interest of or about the Southern Surgical Association. My heart, therefore, is too full to correctly express my feelings. Many of you know that I have said time and again that I am in love with this Association and have always been; every breath I have drawn in regard to it has been one of affection and admiration; and I may say, almost without exception, that I have loved every member of the Association with whom I have in any way come in contact.

Now, gentlemen, you have imposed upon me a sacred obligation. I was not present at the conception of this Association but I was present when it was named, and it is a pleasant recollection to know that I remember very well the elder Haggard, just as well today as I did at the time I became a member of this Association. He was gentle, persuasive, more or less dominating, but of quiet demeanor which carried conviction. He was our first President. And Dr. Davis—what an influence that man had! I cannot begin to name the men who have influenced my thought and my surgical character, to whom I owe my profound thanks, when I look over the names of the members of this

Association. It has always been an inspiration to me, and I feel that I ought not to consume any more of your time except to refer to one or two things which occurred to me last night. Our President, in his beautiful, chaste and eloquent words, not only called my attention to our responsibility at the present time, but none of us can know what may happen before we meet again. The blood of our citizens, perhaps the blood of our sons, will be shed in a foreign land. My patriotism was stirred by that address. I am glad that I came to this meeting because it has given me greater inspiration and greater courage.

Gentlemen, I am unable to say more, but I want each one of us to feel that we will enter the next year with courage, with patriotism, holding up not only the banner of this Association but the banner or our country. I thank you very much for the distinguished honor you have conferred upon me.

22. "A Specific for Every Microörganism," by Dr. Edward H. Ochsner, Chicago, Illinois.

23. "The Use of Bull's Serum in the Treatment of Wounds Infected with the Bacillus Aërogenes Capsulatus Welchii," by Dr. Alexius McGlannan, Baltimore, Maryland.

Discussed by Drs. Jackson, Moore, and in closing by the essayist.

24. "Traumatic Brain Surgery," illustrated by slides, by Dr. John Wesley Long, Greensboro, North Carolina.

25. "Report of Cases: (*a*) Sacral Teratoma; (*b*) Glioma of the Upper Abdominal Region," by Dr. James E. Thompson, Galveston, Texas.

Dr. Hugh H. Trout offered the following resolution:

Resolved, That a vote of thanks be extended to our Committee of Arrangements; to the Honorable J. E. Ingraham, Mayor of St. Augustine, and to Dr. Andrew Anderson, its most respected citizen; to Frank E. Matthews, Secretary of the local Historical Society; to William McAuliffe, Manager of the Hotel Alcazar, and to the *St. Augustine Record* for reports of proceedings.

Seconded and carried.

Dr. Thomas S. Cullen moved that a rising vote of thanks be extended to the retiring President, Dr. Haggard, for the admirable address he delivered last night.

Seconded and carried.

As there was no further business to come before the meeting, on motion the Association then adjourned to meet at Baltimore, Maryland, December, 1918.

ADDRESS OF THE PRESIDENT

THE MEDICAL PROFESSION AND THE GREAT WAR

By William D. Haggard, M.D., F.A.C.S.
Nashville, Tennessee

The Southern Surgical Association, the presidency of which your exceeding generosity most graciously conferred upon me, has for nearly a third of a century been intensely and diligently seeking to perfect methods and men in the science and art of surgery. Each year we have come together with gladness and joy, enthusiastic in the achievements of the hour and intent upon the advancement of our professional ability to relieve our fellow-beings of their pain and disease. Since last we met the alarums of war have been sounded. Our great nation has joined the embattled legions of our Allies overseas in self-immolation for the preservation of the peace and liberties of the world. The impulses of our profession have been kindled, our responsibilities and problems have been inconceivably augmented. Some millions of our bravest and best beloved are consecrating their lives to the stupendous task of making the world free. They will be exposed to the inevitable diseases and multilations which it will be our duty to prevent, to assuage and to repair.

The world is bathed in blood and tears. Its peace has been devastated and destroyed with the pitilessness and terror of an Alpine glacier. This war is a clash between the ideals of democracy and of autocracy. We now know that it was inevitable. The issue is between two political and social

principles, which cannot longer dwell together on the earth. Our enemies believe that might makes right; we believe that right makes might. It takes first rank with the most magnificent events in all history. It will not change maps, but individuals. It will make them free. It is portentous; it is almost sacred in its intent. When the end shall have been attained, it will lift a mighty people upon a higher plane for their development, the great nation, thrice-panoplied, that now faces the world. They know now that the stupendous struggle is for their liberty as well as for that of all the nations on the globe. It is for this that we are sending our brothers, our sons and ourselves to take their stand in this gigantic issue, upon whose outcome the future of all mankind will depend.

Commercial Germany would have made a peaceful conquest of the world, but industrial Germany had no voice. Imperialistic Germany cunningly planned the most unpardonable and deliberately cruel of all wars. They would none of arbitrament. As Van Dyke said, "The Barabbas of war was preferred to the Christ of righteous judgment." Those who loved peace were forced to fight for it or give it up forever.

We must play our appointed part in the world, consecrate ourselves to our principles and policies, put aside self-seeking, distraction and the very peace which we have imagined for ourselves in order to give it unto others. The page on which the history of this most holy of wars will yet be written will be illumined with the great white light of our pure and unstained desire to benefit the cause of humanity.

Was there ever a war before where the victor desired no spoils? Should the lofty disinterestedness of our cause and the superiority of our might give us the victory, there will be no vindictiveness or economic vengeance wreaked upon our enemies. Our loftiest aims and most sacred ideals have been crystallized and formulated by that great seer, that incomparable patriot and immortal statesman, Woodrow Wilson, Prime Minister of the world.

If our nation lives up to its own plighted word it will gain for itself new honor and imperishable fame. If our sacrifice establishes on a big scale and securely those great institutions and opportunities which make men free, then the contest rises to the sublime. Our enemies are not willing to be responsible for this war. Although inaugurating it they disavow their initiative and summon their scientists and philosophers to prove it.

It is not the Teuton or the Oriental who is the enemy of civilization. Militarism is the enemy. From the Straits of Dover to the very gates of the Garden of Eden itself grim-visaged war has raised its bloody head. It is into this "hell of iron" that the youth and flower of American manhood must fling themselves. They must join in the comradeship of arms and the exaltation of spirit with their intrepid and fearless British cousins and with the heroes of clear-eyed France, torch-bearer of the nations.

It was of a poilu that Henri Barbusse wrote: "Each one knows that he is going to take his head, his chest, his belly, his whole body, and all naked, up to the rifles pointed forward, to the shells, to the bombs piled and ready, and above all to the methodical and almost infallible machine guns, to all that is waiting for him yonder and is now so frightfully silent, before he reaches the other soldiers that he must kill. They are not careless of their lives, like brigands, nor blinded by passion like savages. It is in full consciousness, as in full health and full strength, that they are massed there to hurl themselves once more into that sort of madman's part imposed on all men by the madness of the human race." It was a manly young fellow, like one of these, who when brought mutilated to the dressing station by the stretcher-bearers said: "I offered France my life and she took only my arms." A young English soldier mortally wounded was seen suddenly to leap into the air and with his last breath cry out: "Are we down-hearted? No!"

Are the best physicians and surgeons of our country too

skilful or too gentle to be permitted to minister to the fine fortitude or such splendid bravery? No! the faithful sons of Esculapius have never faltered. As Sir Berkeley Moynihan has said: "We are, as a profession, by intellectual descent and by solemn adoption, the heirs of the men who have made our race great and famous." It will always be an inspiration to us to remember that the first American to carry the Stars and Stripes to our stricken Allies after the fateful April 6, 1917, was a surgeon bearing also a Red Cross, a member of this Association, Major George W. Crile. It is a benizon to our profession that the first officer in the uniform of our country to yield up his life was also a physician, Lieutenant Fitzpatrick, who was struck by a shell that exploded while he was standing in the door of the Washington University Base Hospital.

The personal and material sacrifices which our guild have made and are making are not equalled by any other profession or class. We rejoice in its contemplation and take inspiration from the fact that only through the efforts of the medical profession has the prosecution of the war been possible. It would, as our distinguished Fellow, C. H. Mayo, has said, have been terminated long ago through the same causes, which have terminated all wars in the past, through disease and infection.

In the momentous hours of history some individual with transcendent attributes seems to be raised up. The essential to preserving an effective fighting force is primarily vigorous and immaculate sanitation, scientific and uncompromising prophylaxis. Who could have been more opportunely fitted for this task than the man who conquered the deadly and pestilential Canal Zone—the brilliant Southern scientist and knightly soldier—Surgeon-General William C. Gorgas? He has summoned about him many score of our best sanitarians, surgeons, internists and specialists, who have unstintedly given of their time, knowledge and labor. He has sent nearly two thousand medical officers to our needy Allies. He has

builded, equipped and manned hospitals, from 500 to 1000 beds each, in nearly 500 cantonments and camps, caring for cities of from 20,000 to 75,000 soldiers. He has organized courses of intensive special training in the great centers for his medical officers, that, in addition to the training camps for the Reserve Corps, have become the greatest and most comprehensive postgraduate course in the world. He has despatched base hospitals not only for our own expeditionary forces, but out of his abundance has loaned to the other nations, and yet the manifold activities of the Surgeon-General's office, while incomputable, have only begun.

The distressful epidemics that ravaged our armies in the Spanish-American War were deadlier to our soldiers than all the bullets fired in the Antilles. Preventive medicine, especially as applied to armies, has made tremendous progress. Witness our unparalleled feat of mobilizing last year on the Mexican border more soldiers than have bivouacked since the Civil War, more men than belonged to any one command during that sad conflict, more men than were enlisted against Spain; and, instead of marking the border with lines of tombstones, we brought back the 100,000 national guardsmen with a net gain of over 1,000,000 pounds. There was scarcely a case of typhoid fever, and the usual infectious diseases were banished as if by magic. At Chickamauga, in 1898, there were 10,000 cases. During the three years of the present war the British army, with millions of men engaged, have lost only 292 men from typhoid fever. Typhus fever has been denied access to the battleground of the far-flung Western front. We will see to it that it will never gain a foothold in our army camps. Tetanus is almost completely prevented. So far as lockjaw is concerned, the bullet is as harmless as the sting of a bee. And now from the Rockefeller Institute comes another discovery—an antitoxin for the gas bacillus, by Dr. Carrol W. Bull. You will honor him as a Southerner, and I will acclaim him as a Tennessean.

The Medical Reserve Corps now numbers nearly 15,000

physicians who have volunteered and been commissioned. This very remarkable mobilization, in eight months, of over one-sixth of the active practitioners in this country has been made possible by the far-seeing and highly proficient labors of the medical section of the Council of National Defense. It is due to the patriotism and superlative organizing capacity of one of our Fellows, Major Franklin H. Martin, who associated with himself another of our Fellows, Dr. Frank F. Simpson, and these two, with indefatigable effort, beginning nearly a year before war was declared, organized every State and rendered physicians available and effective. This is the first time our profession has been given its due meed of recognition in being honored by representation on the Advisory Commission of the Cabinet, a recognition long merited and signalized by an appointment most felicitous.

Through the further elaborate activities of the Medical Section of the Council of National Defense and the far-reaching organization of the American Medical Association, most elaborate plans have just been perfected by the creation in each State of medical advisory boards to reëxamine certain registrants in the reclassification of 9,000,000 men, which is now beginning. It is not too much to say that the physicians and surgeons were perhaps better organized and mobilized than any other newly created fasciculus of our great structure of preparedness.

Through the endeavors of medical men sanitation has become so perfected that the only danger to the soldier is the bullet, and, if he is not killed outright, the superior methods of treating war wounds deprives them of many of their dangers. The excellent work of Carrel and Dakin in putting antiseptic management of war and industrial wounds on a higher and more wonderful plane of usefulness is a contribution of stupendous moment to humanity.

If medical men have been of indispensable value in this war, who will compute the improvement in the management of every type of injury to the human body? Dr. Crile

declares that "more progress has been made in the surgery of the chest and abdomen, in the treatment of wounds, of infections, of hemorrhage and exhaustion; more knowledge has been accumulated of splints, of apparatus and of every applicable mechanism in the three brief years of war than in the past generation."

Apart from the humanitarian aspects of the war it will in its end-result in the eyes of future generations be regarded, Cleveland Moffett says, "the war beautiful." The French Revolution with all of its terrors quadrupled the scope of civilization; the American Revolution with all of its sufferings was of all wars the most constructive; the Civil War with its bitterness cannot now be but looked upon as essential and evolutional. One cannot be unmindful of the millions of lives already sacrificed, nor of thrice these millions who have been wounded and maimed. In comparison we must consider the lives sacrificed in peace by preventable disease, by unnecessary industrial accidents and deaths, by enforced poverty, by the evils of alcohol, by prostitution and by wanton manslaughter. If, as the result of the supreme and essential sacrifices of this war, we necessarily or voluntarily safeguard human life—men, women, mothers and little children—the saving will exceed the wastage.

Already this war has emancipated men from the slavery of alcohol, the greatest curse and blight upon humanity. The gain in food products will be incalculable. The tilling of the soil will be raised to the nth power, which will make for home building. National safety will oblige essential reforms of our tenement system, our slums; of capital, that it shall not get more than a reasonable and just profit; of labor, that it receive its full and fair reward. It will ensure equal opportunities for women; it will signal the end of fabulous fortunes. The wealthy classes will be quickened into keener appreciation of citizenship. Statesmen will be recruited from men of parts instead of from politicians. In lieu of luxurious indulgence, abstemiousness will be the

fashion. The creed of physical fitness will be embraced, universal military training will probably become effective for physical reasons if not for martial needs.

The social disease which has heretofore been considered the inevitable pestilence of armies is being fought with every imaginable agency—education, recreation, diversion, protection, isolation, prophylaxis, penalties and court-martial. Many thousands of young men will, for the first time on a wholesale plan, be taught the whole truth by all sorts of real men, and purity made a cult, a win-the-war asset. After the war the idea will permeate all strata of society and be a real understandable and livable benefaction. It will disseminate throughout America the practice of personal hygiene by uncounted numbers of young men. The beneficent results to accrue to us and to posterity will almost make the war worth the sacrifice.

When our soldiers reach France it seems that they become exalted with the purpose of victory; they have lived the clean life and believe in it; they have been known to avoid all temptations in the great cities during furloughs. It is army experience that a sober man seldom seeks impure associations. Our American youth will learn reverence for authority, discipline, obedience, immediate and implicit. For the duration of the war the intensive high-minded instruction that will be inculcated into the minds and lives of young Americans will work a veritable physical and moral rebirth of this nation.

As the by-products of industry are the most important, so the fierce necessities of war make many collateral advancements. Great progress in many branches of manufacture have been brought about. We will of necessity make our own dyes and our own chemicals of all sorts. To effectively educate 500,000,000 people in the brotherhood of mankind could not have been accomplished without the lessons and results of this war, costly as it is.

When the carnage has ended the world will have drained itself well-nigh dry. Much of its best and most precious blood will be spilled. Its liquid capital will have been used up. It will be the privilege of this nation to bind up the war's wounds and to slake the white-heat of hatred—to be in the vanguard in the colossal work of reconstruction and of rehabilitation. Enormous problems will present themselves when our army disbands. Years will be consumed in demobilization. Restoration of our soldiers to the pursuits of peace in an equitable manner will be a nation-broad duty. The conduct of even so gigantic a war as this is comparatively simple to the colossal task of making the world over again when it shall have ended. But the reëducation of the crippled, the maimed, the sightless and the salvaged after the war is a voyage across uncharted seas into another world. No longer will we attempt to salve the wounds of heroism with alms or allow a mutilated patriot to eke out a pitiful existence as best he may; rather will it be our splendid aim to reëducate that unfortunate so as to give him a trade, or a profession more lucrative, more independent and ennobling than he had before.

For nearly a century and a half our nation has wrought into permanence those principles, both of government and of right, for which the philosophers have dreamed since the renaissance. Our federation of States is now the greatest, the wealthiest and the most powerful exemplar of democratic institutions in Christendom. It has been shown that the only way to make the world safe and secure is to entrust it to accredited representatives of the people and not confide it to dynasties or diplomats, however great.

This is a war for the creation of a new international world—a war for a new intranational world. Human liberty, justice and the honorable conduct of an orderly and a humane society are the ideals of life which must prevail. A combination of events has forced the United States into the position

of leadership. We have demonstrated that race antagonisms tend to die away and disappear under the influence of liberal and enlightened political institutions. Consider our large Babel-tongued population, all living in peace and harmony, and as years pass they are melted in the crucible of democracy and are molded into Americans with all the strength and freshness of a nascent recreation. Our democratic institutions have shown their ability to amalgamate and to emancipate every type of human being which has thus far come under our flag. It is the alchemy of the nations. Why should not each nation in Europe establish for itself a place in the sun of unity which may come when the war clouds have been swept away? Who knows but coming out of this dread conflict in which the civilized world has been plunged will issue, as Nicholas Butler Murray has said, the United States of Europe? In the end each of the nations of the earth will deposit in a world's federation some portion of its sovereignty for the perpetuation of peace and the furtherance of good-will to all.

It has been touching to see in this country the spirit of generosity, of sympathy for the afflicted, the distressed and the stricken in the uttermost parts of the world. Although denounced as get-rich-traders, the American people have been lavish with their millions and have given their lives and their endeavors to carry food, clothing and succor to the starving Belgians and the other desecrated nations of bleeding Europe. We have played the Good Samaritan on a huge scale. Never in all history has there been such a generous outpouring of tenderness to those who needed help, irrespective of race, station or belief.

In this beneficent work the representatives of the healing art have had their share. For the Southern Surgical Association, to its lasting honor, your President wishes to record that out of 200 members, 50 of whom are disqualified by age or obvious disability, over 80 have given their services to their country. They are soldiers, brave and fearless, yet they are

gentle and harmless as doves. That great anatomist, physician and author, Oliver Wendell Holmes, has beautifully said of our ministration:

"As life's unending column pours
  Two marshalled hosts are seen;
Two armies on the trampled shores
  And death flows black between.

One marches to the drum-beat roll
  The wide-mouthed clarion's bray
And bears upon its crimson scroll—
  Our glory is to slay.

The other marches in silence by the stream,
  With sad, yet watchful eyes,
Calm as the patient planets' gleam
  That walks the clouded skies.

Along its front no sabers shine,
  No blood-red pennons wave,
Its banner bears the single line—
  Our glory is to save."

SOME UNUSUAL SURGICAL EXPERIENCES

By Albert Vander Veer, M.D.
Albany, New York

We have all kinds of research laboratories, yet I have sometimes wished there might be an institution for research observation. My experience has brought me in contact with a class of cases, such as foreign substances in the esophagus, trachea, bronchi, also the rectum, requiring immediate treatment, a few of which I would like to present to complete a promise to give you a paper for this meeting. The conclusions I refer to in regard to treatment, in the past and present, may elucidate a little more clearly these preliminary remarks.

Master G. C., aged seven years, a patient of Dr. Van Alstyne's, while handling his father's lead-pencil, loosened the head, and during the act of laughing inhaled that portion. The patient coughed a little at first, but not constantly, and was restless during the night. He was brought to the office the next morning, a distance of some fifty miles, breathing with considerable difficulty. On examination no respiratory murmur could be heard over the right lung and no marked dulness on percussion. It was believed the foreign substance had lodged in the right bronchus, possibly just below the bifurcation. He was admitted to the Albany Hospital, an anesthetic carefully given, a rather rapid tracheotomy done and curved forceps passed down into the bronchus, with some trouble, where the foreign substance could be felt. The

first attempt at securing it did not succeed, but the second caught the edge and the tip was removed without any difficulty. Rather violent coughing followed. Prompt withdrawal of the anesthetic was made, and after some escape of mucus into the incision the patient became conscious; he was easily calmed and earnestly appreciated being told the pencil-tip had been removed. He made a rapid recovery and the trachea healed promptly.

I have great respect for the skilled specialist who can so deftly handle the esophagoscope and bronchoscope, but I am bound to say that these instruments in the hands of an unskilled operator produce more harmful results than follow the clean operation.

It is true that this case presented before the use of the *x*-rays. Today, with the use of the latter, the location might have been more definitely determined; but as we cannot all become experts in the employment of the instruments to which I have referred, and as these cases must be attended to promptly, I believe that tracheotomy is not to be excluded in affording immediate relief—that less traumatism results than from the experimental use of unfamiliar instruments.

I have seen fatal results from the toy mouth balloon, with immediate suffocation, yet, withal, some remarkable recoveries, from inhaling into the bronchi foreign substances such as a kernel of corn, a bean, a bit of straw, a clover-leaf, a peanut-shell or portions of the peanut itself. I have seen some of these substances, particularly a locust-pod, expelled as soon as an incision was made in the trachea, when a violent fit of coughing took place; have also observed the abscesses that would result from foreign bodies, one, two, three or four months after inhalation, in consequence of a large enough bronchus connecting with the cavity in the lung, when with a sudden cough and expectoration of pus the material would be discovered. I have never operated upon an abscess of this sort from the exterior portion of the chest. Metal substances, like a tack or possibly a small nail,

as I have observed in many instances, when inhaled, will become encysted; but any substance that will macerate is apt to be expelled in the manner I have described. The most difficult and quite impossible material to reach is a cork or some such material. It cannot be grasped by any ordinary instrument, and I do not think the bronchoscope has demonstrated reports of a sufficient number of successful cases to warrant much comfort; yet I believe it is in these cases such instruments are to prove of great value.

It is quite interesting to note how persistently a foreign body will remain permanently between the cricoid cartilage and the bifurcation, moving up and down, thus giving the child a chance to breathe, although lodged at that point. One case like the following will serve as an illustration:

A girl, aged seven years, had inhaled a bean, which was readily recognized located in the respiratory tract. At times she would have severe attacks of coughing, but the substance was not expelled. The patient was brought to the hospital, where I made no effort to remove it from above, but did an immediate tracheotomy. The bean was easily located and removed, the patient making a good recovery. I saw her several years later, when there was no appearance of scar, no constriction or evidence of any traumatism to be noted as the result of the operative intervention.

Foreign substances taken into the esophagus or the respiratory tract, and becoming lodged and partially covered by the mucous membrane, may sometimes lead us astray regarding the symptoms we gather from the patient.

The following case is remarkable in many respects, and today, by means of an examination with the bronchoscope, would lead to a more correct diagnosis:

Miss E. N., aged twenty years, in 1900, while dressing her neck insisted she had swallowed a black-headed pin about two inches in length. She complained of distress in swallowing. The symptoms presented with such urgency I was induced to do an esophagotomy at the end of twenty-four

hours. This was performed without any difficulty, but I was unable to find the pin at a point where it could be removed, although I could feel it through some thin tissue. I was obliged to admit it was outside of the esophagus and not safe to attempt any further operative intervention. The patient made a good recovery, having a cough that was prolonged for quite a while, but which eventually disappeared. In the spring of 1916, during a severe paroxysm of coughing, Miss N. expectorated quite freely and noticed in the material what she thought was a bristle, but upon a more thorough examination the substance was found to be the lost pin. The head had disappeared, and in handling it, thinking it a bristle, she broke off the little point, which was plainly to be observed. Clearly here was an incomplete diagnosis, for undoubtedly at the time she inhaled this into the bronchial tube instead of swallowing it. In examining her neck there is no evidence of any scar tissue to be observed; she has had thorough use of the neck muscles and no trouble in swallowing. The bronchoscope would have been of great value in this case.

It has been a wonder to me that animals do not oftener die from some foreign substance lodging in the esophagus, as they eat all kinds of food. My attention was once called to a pet cat which was unable to swallow anything, had grown very much emaciated and finally died. An autopsy was made and a piece of fish-bone found in its esophagus, very strong, nearly the size of a nickel and with sharp edges.

The case reminds me of one occurring in the practice of a colleague years ago:

Mr. B., after partaking of a meal of roast beef, complained of difficulty in swallowing and entered the hospital for relief. With the metal probang the doctor could locate a foreign substance in the esophagus. On consultation the use of the umbrella bristle probang was advised, but on doing so a sudden gush of arterial blood presented and the patient died of acute hemorrhage. Autopsy revealed the arch of the aorta had been punctured by a portion of bone, very much

like the fish-bone that had been sliced in the cutting of the beef, and caught in the probang. Would the esophagoscope have averted this accident?

My observations permit me to report one or more cases of foreign bodies, such as chicken or other kinds of bone, lodging in the esophagus, becoming buried beneath the mucous membrane and eluding search when an esophagotomy was done. At the present time this could scarcely occur by the intelligent employment of the esophagoscope. We certainly profit by research operation, and had my case of a pin in the bronchus had the benefit of the use of the bronchoscope an esophagotomy could have been avoided.

Foreign bodies in the rectum have always been a source of considerable thought, and just how to remove them promptly at one time gave me some little annoyance. At first they may easily be felt, but not so readily grasped, and I early demonstrated in my own practice that it was safe to dilate the sphincter sufficiently so that a small-sized hand could be inserted, the substance being grasped in that way. It is well known that these foreign bodies have a very energetic way of getting up above the sigmoid flexure into the descending colon. I have come in contact with an ink-stand and such odd articles, but in the case I have in mind the foreign body was pushed into the rectum and caught in this manner. A wax candle such as was used in the passenger cars of years ago is most remarkable. The engineer of the train suffered from eczema about the anus, with pruritus ani, and was in the habit of securing some relief by rubbing the parts with the end of a candle. On one occasion, while doing this, he suddenly became faint, and sat down on the candle, which immediately disappeared in the rectum. It was a difficult matter to reach this foreign substance and remove it, but it was finally accomplished, with some considerable trouble, by dilating the sphincter, when the candle dropped into view.

In my earlier years of practice, when called to far distant places in the country, often inaccessible mountainous regions,

when the opposition to moving the patient to the hospital was very great, not infrequently I have been obliged to operate at once, leaving the case in charge of the family physician, with a line of treatment as practicable as possible for those in attendance.

April 22, 1892, I was called to see and to operate upon Mr. S. P. G., aged eighty years, living with his wife and two elderly daughters. He had suffered for more than two years from an irritable bladder, with much distress in passing urine. All symptoms were indicative of stone, which was located by means of the Thompson sound. It seemed quite impossible to give him much preliminary treatment, and an immediate operation was demanded. He had an enlarged prostate, was in rather feeble condition, so that the operation that could be performed the quickest and produce the most satisfactory results commanded my attention. Under ether I did a suprapubic cystotomy, removing a good-sized stone. I left a No. 12 English rubber catheter in for drainage and the wound was kept in good condition by the intelligent care of his family physician and the aid of his daughters. The wound healed moderately, but little restoration of the flow of urine through the urethra. He made a rather remarkable recovery up to this point. In consultation with the doctor I suggested permanent drainage, keeping it in from above, and had two silver tubes made, which the daughters kept in good condition. By plugging the tube it was soon found that the bladder would hold fully four or more ounces, then evacuating by the use of a small catheter through the tube kept the parts in a very clean state. At times he would pass a distinct stream through these silver tubes. From time to time, however, the bladder was irrigated through the catheter and kept in very good condition. Evidently there was no invasion of the pelvis of either kidney and the urine remained quite normal. Under this treatment he was made very comfortable and lived for a period of five years longer.

Possibly at the present day we would have done a prosta-

tectomy and restored the function of the urethra. I have occasionally used this same form of treatment with other patients who lived at a distance and could not have a more radical operation. Today most of these patients would come to the hospital for more modern, up-to-date early treatment.

I hope I may be pardoned for referring to a case bearing upon abdominal surgery and injury to both the esophagus and trachea which came under my observation and treatment in my earlier years of practice and illustrating the persistency with which prisoners will attempt suicide. I have observed when not successful that in a second effort they seldom make a third.

Mr. E. B., aged thirty-eight years, an inmate of the Albany Penitentiary—on a thirty years' sentence for counterfeiting—July 10, 1878, with suicidal intention, made an incision in his abdomen of some length, tried to divide the right carotid and then severed the left brachial artery one or more inches above the elbow. From this last wound the bleeding was very profuse. I cannot call to mind when so desperate an attempt was made and so much accomplished in the intention. The knife used was made from a steel spring used in the shank of a lady's gaiter. The act was committed in his cell at night and he was not discovered until the next morning. The wound in the abdomen was seven inches long, from a little to the left of the umbilicus to the ensiform cartilage, through which protruded the stomach, the large and small intestines, with the omentum, which was gashed in several places; a distinct amount of fecal matter was on the skin, the viscera being further cut after protrusion. The intestines were cold and dry and had fuzz from the blanket sticking to them. The parts at first were wiped with a dry handkerchief and returned after an hour. The wound was then closed with eight interrupted sutures and broad bands of plaster applied around the trunk. Bleeding having ceased, other wounds were brought together with plaster only.

The neck wound was quite superficial. He was kept under the influence of morphin and after forty-eight hours removed to the prison hospital. At the end of four days the sutures were removed, the wounds having nearly healed by first intention, with about one inch of abdominal wall still open, but were healed at the bottom when I saw him July 27, through the kindness of Dr. Haskins, the penitentiary surgeon, and who then gave me permission to use the history of the case. There was very little pain and no evidence of peritonitis at any time. The pulse was always below 100; temperature never notably above normal. Morphin was discontinued on the eleventh and bowels moved voluntarily on the fourteenth day. The wound in the left arm was one and a half inches long, over the lower third of the brachial artery, but the vessel was not in sight. When I saw him a firm, cylindrical mass occupied the place of the brachial artery, where it was wounded, pulsation being felt two inches above. Pulsation ultimately became perfectly plain in the left radial and the patient returned to duty November 1.

Some two years later, with two small knives, he made another attempt at suicide, apparently using a knife in each hand, making a deep incision in his neck, cutting across the trachea completely down through the esophagus, severing the posterior wall, but not injuring any important vessels, except the inferior thyroid on the left side, from which he bled profusely. I saw him a short time after the attempt was made, and a more ghastly wound I have seldom seen. He was breathing out through it, forcing the blood and mucus over his chest. When completing the examination and ligating the inferior thyroid the steps of the operation for repair became quite clear. With catgut sutures I brought together the ends of the esophagus and with silver wire the rings of the trachea, then closed the severed parts with layers of catgut and drainage. With kind and patient nursing he made a good recovery. When all was over and he was about

to return to his work in the shop he said to me very earnestly, "It is not possible for me to die by my own hands, and I shall never make another effort." He always maintained he considered himself innocent. He was a superior workman, had engraved the counterfeit plates and sold them for a certain sum, but never passed a dollar of the spurious money, nor was he responsible for offering it to the public. His behavior afterward was so excellent he was later pardoned and lived a very simple, quiet life with his family, ultimately dying of tuberculosis. At the time the case attracted a good deal of attention, and, later, when aseptic surgery was introduced I have often thought of this and other cases when the surroundings seemed so utterly inconsistent. In Mr. B.'s case death would ordinarily have occurred from hemorrhage, shock or peritonitis. The completeness of the division of the brachial artery was evident in the amount of hemorrhage; while it is possible, as he was lying on his left side, the elbow became flexed, the forearm pressing against the arm and checking the flow. (There is a chance for some doubt, the circulation returning rather quickly.) The warmth of the weather and being covered by a blanket prevented shock from exposure; the protrusion of the abdominal contents perhaps was not soiled, as was thought at the time. The intestinal wounds being inflicted after protrusion, possibly no fecal matter entered the peritoneal cavity, and from long exposure all oozing had ceased before the return of the parts; the wounds being closed by exudation, many conditions on the part of nature favored his recovery as well as preventing peritonitis.

In doing my abdominal work I have always had a fear regarding the possible breakage of a glass drainage tube and was led to make use of the aluminum ones; however, I eventually returned to the former, as they could be sterilized with a greater degree of ease and certainty.

There is much anxiety in a hurried operation when conditions seem to demand it, not only regarding the surround-

ings of the patient but, more especially, the serious state they may be in. I have been called late at night to distant points, perhaps operating during the night, or, under the constraint of knowing you have only a certain amount of time before you desire to reach the train that will take you home within a reasonable period, and the anxiety following some cases, as to their progress afterward, had been a source of much worry to me, as illustrated by the following case:

Mrs. A. C., aged thirty-five years, married, usually in good health, in November, 1907, became very ill with marked symptoms of acute appendicitis. I was telegraphed for in the evening, and on reaching her home late, taking with me my assistant and a trained nurse, found her symptoms very urgent. An immediate operation was demanded, and not having any too much time, in consequence of the desire to return on the earliest possible train, the operation was done quite rapidly. A nasty, gangrenous appendix was found and a glass drainage tube inserted; the wound was properly dressed and directions were given the nurse regarding the treatment. The patient progressed favorably and drainage lessened so that at the end of four days, on hearing from the nurse by telephone, it was thought well to remove the glass tube and insert a smaller one. On removal the nurse was very much distressed to note that the end was irregularly broken, and at once telephoned me of the situation and sent me the tube the next day. Naturally, I was considerably perturbed, thinking of the possibility of the glass having done some harm to the bloodvessels, and advised the nurse not to acquaint the husband or any member of the family regarding the matter. The patient made a good recovery but the fecal fistula weakened the abdominal walls, so that a few months later it became necessary to do another operation. In doing this I made a diligent search for any possible pieces of glass, but failed to find the same. The patient convalesced nicely from the second operation and has never had any untoward

symptoms. In thinking this case over I am of the impression the tube was broken at the time the nurse handed it to me, but in my haste to complete the operation and get my last midnight train I overlooked it. Some of you may call to mind the state of anxiety I was in when informed of such a condition, and this case gave me more worry than a week of hard work. Perhaps it may be of interest for me to make the following statement:

The husband, a county official, was found to be a defaulter, and while he lived in fine style, the house being commodious and well furnished, with a splendid library, yet I never received any remuneration for my visits or the operations. He was convicted, sent to prison and his ill-gained wealth vanished, leaving his wife absolutely penniless.

About 1870, in my early years of practice and before we were doing abdominal work to any extent, I was called by Dr. W. H. Bailey to see a case which had been diagnosed as general peritonitis.

Mrs. B., aged about thirty-two years, married, and with living children. Patient was ill less than a week. Her abdomen was distended and exceedingly tense and condition desperate. She pleaded so earnestly for relief that I resolved to pass a small trocar in a zone of great resonance, just below the umbilicus, in the median line. The escape of gas was most pronounced; the cannula was left in for some time and the patient made a complete recovery. Soon after this, as my consultation work increased, I saw too many of these cases for my own comfort. At that time they were called by many of the physicians "idiopathic peritonitis," but they were the eleventh-hour consultations so far as I was concerned. It was impossible to determine the exact cause of the trouble in many instances. These cases have at times haunted me, even up to the present hour, in thinking how little we were then able to do for them. The use of the aspirator was tried at times, but with no permanent effect. The treatment forcibly recommended by Dr. Alonzo Clark, of giving opium

to patients, was all that was done at the time aside from the use of enemas, the latter too often doing but little good. The contrast between them and now is so great, especially the technic in the performance of an enterostomy, that the middle-aged and younger practitioners of today little realize the struggles we met with in our early abdominal work.

May I say a word in expressing my appreciation of the excellent work this Association is presenting to our fellow-laborers throughout this great country of ours? It has given me great joy to go over the last volume of its *Transactions*. What a treasure for the earnest, up-to-date worker in our professions! We have a right to congratulate our officers upon the splendid ending of another year of successful effort.

THE NEED AND VALUE OF BIOPATHOLOGICAL STANDARDIZATION

By William Carpenter MacCarty, M.D.
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In reviewing the present status of tissue pathology and its terminology in relation to clinical medicine, it seems that there has been a stationary period of about twenty years in which it has not rendered the greatest degree of efficiency.

It is possible that this period of quiet had its origin in the fact that tissue pathologists were dealing with end-results, as seen at autopsy plus postmortem changes, and the imperfections of methods of fixation, all of which probably misrepresent the facts as they exist during life. The resultant conception made upon both pathologists and clinicians is somewhat comparable to that obtained in the study of systematic botany from dried, pressed specimens as compared with that derived from growing specimens in the field, or the conception of birds which is derived from stuffed skins. Such artificial and unnatural methods of study, however, certainly have their place in science and should not be condemned. Without them we should never have reached our present knowledge; but their usefulness as makeshifts is losing its value in the presence of newer methods and observations in pathology, clinical medicine and general biology.

The second probable reason for the quiet of investigation in tissue pathology may be the fact that bacteriology, with its closely related sciences such as immunology and serology,

has made its greatest advances during the last twenty-five years and has drawn men of vision, imagination and initiative away from tissue pathology.

The pupils of such great teachers of tissue pathology as Virchow, Cohnheim, Ribbert, Rokitansky, Chiara, Orth, Borst, Welch, Councilman, Hektoen, Leconte, MacCallum, Adami and others have become immunologists, bacteriologists, serologists, sanitarians and cancer experts.

The field of tissue pathology has been practically deserted. Hospitals and teaching institutions are beginning to realize the great dearth of efficient men to carry on tissue investigations for clinical diagnostic purposes which they realize are absolutely necessary for efficient medical and surgical practice.

The following facts are examples of the necessity of expert microscopic tissue diagnosis to the clinician, surgeon, and pathologist:

I.—FROM A CLINICAL STAND-POINT

	Cases.	Per cent.
Cases registered, July, 1917	4752	
Operations	1699	35.0
Surgical specimens	1046	22.0
Surgical specimens removed for diagnosis	179	3.5
Necessary microscopic diagnosis	208	4.3
Cases operated upon	..	35.0
Cases producing surgical specimens	..	22.0
Cases producing specimens for diagnosis	..	3.5
Cases which need microscopic diagnosis	..	4.3

II.—FROM A SURGICAL STAND-POINT

	Cases.	Per cent.
Cases registered, July, 1917	4752	
Operations	1699	
Surgical specimens	1046	
Specimens removed for diagnosis	179	
Necessary microscopic diagnoses	208	
Operations which yield specimens	..	61.0
Operations which need microscopic diagnosis	..	12.0

III.—FROM A PATHOLOGICAL STAND-POINT

	Cases.	Per cent.
Cases registered, July, 1917	4752	
Operations	1699	
Surgical specimens	1046	
Necessary microscopic diagnosis	208	
Specimens removed for diagnosis	179	
Specimens removed for diagnosis needing microscopic diagnosis	149	
Surgical specimens which need microscopic diagnosis	..	19.8
Diagnostic specimens	..	83.0

IV.—PERCENTAGE RECKONED WITHOUT APPENDICES, GALL-BLADDERS AND OVARIES

	Cases.	Per cent.
Surgical specimens	1046	
Appendices, gall-bladders, and ovaries	321	
Surgical specimens minus appendices, gall-bladders, and ovaries	725	
Necessary microscopic diagnoses	207	
Necessary microscopic diagnoses	..	28.0

These figures clearly indicate that clinical medicine needs tissue experts in order to render efficiency to the patient.

Many clinicians and surgeons have the impression that they can make their own pathological diagnoses, but this is but an impression which is rarely, if ever, corrected by them statistically. Such impressions must be filled with error in the light of the fact that tissue experts with very extensive experience can diagnose only about 75 per cent. of all specimens grossly; and if they rely upon old standards of microscopic diagnosis, they find themselves face to face with a possibility of microscopic diagnosis in only about 95 per cent.

The clinician's clinical diagnostic error is usually thought of as being about 5 per cent. As a matter of fact, in such a superficial organ as the mammary gland his actual error is from 2 per cent. to 26 per cent., when he makes positive diagnoses. His apparent error, by which is meant that error in pathological terminology which does not affect the patient, is 8 to 50 per cent.

His natural acuity and honesty have led him to avoid clinical error in from 1 to 57 per cent. of different mammary conditions by utilizing such terms as tumor, nodule, mass, growth, benign (?), malignant (?) or by making no diagnosis.

When he utilizes such a term as carcinoma (?), for example, which he does in 8 per cent. of his cases (1800), his guess is correct in 57 per cent.[1]

These figures represent the condition of diagnostic efficiency in one organ which compare favorably with his efficiency in the diagnosis of many conditions in other portions of the body, which will be published at some future date.

The main factor in which the writer is especially interested at this time, however, is the terminological inefficiency represented by the clinician's attempt to utilize detailed pathological terminology for clinical purposes. His code of communication of ideas has an inefficiency of from 1 to 57 per cent., which is too large to be scientific, although when utilized with the help of tissue pathologists may not prove inefficient in so far as the patient is concerned, because what the clinician and surgeon really want to know, in the breast, for example, is whether the condition is benign or malignant, operable, inoperable or non-operative. Clinicians have been led to believe, however, by pathologists that conditions are either benign or malignant, which is only theoretically true.

The theoretical line of demarcation is similar to the one dividing two pieces of property: it has never been seen. The fence, be it 1 inch or 3 feet thick, is the practical line of demarcation, and so it is in pathology; there is a practical, histological, biological and clinical line which was first described before this Association in 1908.[2] Coincidentally with this description a new observation was made relative to the role played by regeneration in the life history of tissues and the recognition of the process of regeneration as a protective process of cells against an antagonistic environment.

It was later recognized that this reaction of cells is similar to the protective activities of all living matter.

It was seen that during the structural and functional evolution of multicellular organisms, certain phenomena occurred. Specialization and differentiation of cells were coincident with a diminution of power of reproductivity which in order to preserve such cells or tissues in the presence of constant or periodic destruction must of necessity be carried on by reserve cells, the function of which is tissue reproduction, hyperplasia, and differentiation.

The reaction of these reserve cells (textoblasts) represented, histologically, hypertrophy, hyperplasia and migration, which biologically mean hyperactivity, reproduction and change of environment, and clinically mean benignancy, an indeterminate condition, and malignancy. There is no other phenomenon which is of significance in association with hyperplasia, *i. e.*, attempt at tissue differentiation, which sometimes occurs in the expansive and migratory conditions of hyperplasia. It is well known that the so-called malignant cells (migratory hyperplasia) attempt differentiation when they arrive in an environment which is favorable to such a change.[3]

It is the lack of this phenomenon, in the so-called indeterminate stage, which prevents the expert microscopist from prophesying clinically just what will occur. How can he read in undifferentiated cells of living structures just what they intend to do?

It is this stage which has produced confusion among pathologists and has caused some to call certain conditions malignant and some to call them benign. It is the stage of cytological activity which makes the differentiation of some chronic inflammatory conditions from malignant conditions difficult—in fact, impossible.

It is the biological significance and importance of regeneration with its stages which the pathologists who were dealing with dead tissues and end-results failed to recognize.

It is well known that all but a few tissues are capable of regeneration, and in the case of most tissues the reserve cells are recognizable.

The regenerative neoplastic conditions were originally described in the following terminology:

Primary (restauro-) (benign) Secondary (expando-) Tertiary (migro-) (malignant)	Adeno- Cardiomyo- Chondro- Endothelio- Erythro- Fibro- Glio- Leiomyo- Leuko- Lipo- Lympho- Melano- Myo- Myxo- Neuro- Osteo- Perithelio- Pilo- Poly- Rhabdomyo- Etc.	Cytoplasia or neoplasia.

This conception has been and is applied efficiently to tissue diagnosis from a clinical stand-point.

Although it is applicable to regeneration and neoplasia, it is not representative of all the visible biological phenomena which occur in tissue reaction, and is not therefore complete from the stand-point of either the clinician or pathologist, although it has formed a basis for study, correlation and description of all of the other phenomena.

There are six fundamental reactions which manifest themselves, microscopically or macroscopically:

1. Cytolysis.
2. Atrophy.
3. Hypertrophy.
4. Neoplasia.
5. Differentiation.
6. Inflammation.

There are seven conditions involved in the basis of the newer conception of pathology and its terminology:

1. Type of cell.
2. Normal activities of cells.
3. Biological reaction of cells to antagonists in their environment.
4. The degree of reaction of cells.
5. The gross tissue manifestations of cellular reaction.
6. The duration of reaction.
7. The antagonists which caused the reaction.

There are at least 28 different kinds of cells which form human differentiated tissue which constitutes the human organism:

Audito-	Cyte.
Adeno-	
Cardiomyo-	
Chondro-	
Endothelio-	
Epithelio-	
Erythro-	
Fibro-	
Glio-	
Gusto-	
Leiomyo-	
Leuko-	
Lipo-	
Lympho-	
Melano-	
Myo-	
Myxo-	
Neuro-	
Odoro-	
Osteo-	
Perithelio-	
Pilo-	
Poly-	
Rhabdo-	
Sebo-	
Tactilo-	
Tendo-	
Visio-	

There are three degrees for each reaction:

1. Cytolysis:
    1. Destruction of the cellular wall.
    2. Destruction of the cellular wall; destruction of the nuclear wall.
    3. Destruction of the whole cell.
2. Atrophy:
    1. Reduction of the cytoplasm.
    2. Reduction of the cytoplasm and nuclear plasm.
    3. Reduction of the whole cell.
3. Hypertrophy:
    1. Increase in size of the cytoplasm.
    2. Increase in size of the cytoplasm and nuclear plasm.
    3. Increase in size of the whole cell.
4. Neoplasia:
    1. Hypertrophy of regenerative cells plus presence of differentiated cells.
    2. Hyperplasia of regenerative cells plus absence of differentiated cells with or without partial differentiation.
    3. Hyperplasia of regenerative cells plus migration with or without partial differentiation.
5. Differentiation:
    1. Complete normal grouping and normal morphology of tissue cells.
    2. Incomplete normal morphology of tissue cells.
    3. Partial grouping of cells according to normal grouping.
6. Inflammation:
    1. The cardinal signs of inflammation (rubor, tumor, calor and dolor).
    2. Rubor, tumor, calor and dolor, plus ulceration.
    3. Rubor, tumor, calor and dolor, plus pus.

The biological reactions sometimes manifest themselves grossly as:

1. Circumscribed conditions.
2. Non-circumscribed conditions.
3. Papillary conditions.
4. Polypoid conditions.
5. Cystic conditions.
6. Ulcerated conditions.

They occur in relation to time as:

1. Acute conditions.
2. Chronic conditions.

If these facts be correlated, they arrange themselves in the following manner which gives a plastic but definite terminological key to most of the pathological conditions which we find in tissues.

Acute chronic	Circumscribed Non-circumscribed Papillary Polypoid Cystic Ulcerated	Primary Secondary Tertiary	Audito- Adeno- Cardiomyo- Chondro- Endothelio- Epithelio- Erythro- Fibro- Glio- Gusto- Leiomyo- Leuko- Lipo- Lympho- Melano- Myo- Myxo- Neuro- Odoro- Osteo- Perithelio- Pilo- Poly- Rhabdo- Sebo- Tactilo- Tendo- Visio-	Cytolysis. Atrophy. Hypertrophy. Neoplasia. Differentiation. Inflammation.

The clinician can recognize positively only those conditions which are underscored, although he might possibly recognize some of the conditions under the question marks.

The time does not permit a detailed elucidation with examples of the application of this conception. Although perhaps not yet perfect and complete, it offers a simple conception with a simple code of communication; it is histologically, clinically, biologically and scientifically more efficient than the chaotic, extravagant, inconsistent, empirical and clinically inefficient conception and terminology of tissue pathology which has come down to us from that great and essential period of observation and description which shall ever be remembered in association with the great masters of pathology who had a scientific strength of conviction which was great enough to break away from the mystic empiricism of the ages which had preceded them.

In conclusion, it must be stated, in the presence of this body of progressive surgeons of America, that this new conception, as it exists, was not possible without a study of early conditions and processes which was made possible by what might be called prophylactic operative surgery as it is being practised in association with expert microscopy.

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DISCUSSION

Dr. Thomas S. Cullen, Baltimore, Maryland.—I do not think a paper of this importance should be allowed to pass without being discussed. I desire to express my appreciation of the work that Dr. MacCarty has done. It is a splendid thing to have constructive criticism. The older I become the more I feel that all of us should have a fundamental knowledge of pathology. The more we look into pathology the more we realize that the classification should be simplified and crystallized in order to avoid confusion.

I feel that this Association should emphasize most emphatically that every man who is to make surgery his life-work should have a thorough groundwork in pathology.

I personally wish to thank Dr. MacCarty for the splendid work he has done. I am, of course, not capable of expressing any definite opinion with regard to the new classification he has presented, as I have not had the opportunity yet of using it.

Dr. J. Shelton Horsley, Richmond, Virginia.—I merely wish to say that I think we all owe a debt of thanks to Dr. MacCarty for giving us a simple classification of tumors and neoplasms, making the laboratory end fit in with the clinical end. It is a source of personal comfort to have the division of primary, secondary and tertiary hyperplasias in breast tumor, and, while the classification given may not include all of the conditions we find in our surgical and pathological work, certainly it fits into the clinical classifications. It seems to me, what we need is to get the laboratory and the clinical man into closer touch.

There is one thing in which there is great room for improvement, and that is the usual teaching of inflammation. I have never been able to see the logic of teaching inflammation in connection with clinical surgery, as has been taught for hundreds of years by the laboratory man; namely, that inflammation is a process which is distinguished by heat, swelling, pain and redness, and that inflammation can be caused by bacterial, chemical or mechanical agents. If you cut your finger, it heals by first intention; the process is a simple one without complication. There is hyperemia of repair which is necessary; but, if germs get in, there is a different process of healing. The two processes

are entirely different, yet both are called inflammation. If Dr. MacCarty can in his classification bring out the difference, showing that hyperemia is necessary for a time, and is responsible for the redness, heat, swelling and pain, but is a constructive process, while infection with bacteria is a complication and a destructive process, it would be a great advance.

Dr. MacCarty (closing).—I appreciate the remarks that have been made. I did not anticipate any discussion because, so far as I know, men are not familiar with this conception because it is too new. I have kept it in abeyance for years, working it over and over. This classification is the result of several years of gradual evolutionary work.

In regard to inflammation, I have purposely put the word inflammation in this classification for the benefit of the clinician. Inflammation means to me any kind of reaction, but in order not to be too radical I left it in. It makes no difference where the inflammation is. The cell reacts in four different ways to an antagonistic force. It either becomes destroyed, or becomes hyperactive, or it reproduces itself, or it migrates. If these are not reactions, I do not know what they are. Inflammation is an evidence of localized conflict between antagonistic things and tissue cells, and the fundamental principles of reaction are the same for all living matter, whether human cells or not. They are either destroyed, they become hyperactive, they reproduce themselves, or they migrate. It is just as true of the cells of our body as it is of our soldiers.

THE DEVELOPMENT AND PERFECTION OF THE INTERPOSITION OPERATION FOR PROLAPSE OF THE UTERUS AND BLADDER

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The incentive which finally resulted in the perfected interposition operation for the cure of prolapse came through the work of Arx, Sanger and others, who abandoned denudation of the vaginal walls and attempts to secure fascial support for cure of cystocele, and, instead, split the anterior vaginal wall in the median line, separated the bladder from these flaps and secured it to the anterior wall of the uterus. The excess of flap was necessarily excised, and the result was a great advance in the treatment of prolapse of the bladder. It was but a short time after this that operators found certain difficulties in applying this method, as inevitably occurred when the uterus was retroverted or prolapsed. It is highly probable that Mackenrodt found that vaginofixation was a logical method of disposing of both conditions, as he shows in his article in *Centralbl. f. Gynäk.*, 1893, xvii, 665. The suggestion of using the uterus to prevent and cure prolapse certainly followed promptly after the publication of Mackenrodt's paper, although we are inclined to the opinion that Freund deserves the credit for the original thought. His (Freund's) first proposition was to turn the uterus into the vagina from behind through an incision into the cul-de-sac of Douglas.[1] Fearing that the cervical canal might become

[1] See Döderlein and Krönig.

occluded, he formed a second opening through the fundus for the escape of mucus or menstrual flow and completed the operation by carefully suturing the uterine body to the vaginal walls. This attempt of Freund did not attract universal attention, as many of the clever suggestions of this brilliant man had previously done, and, furthermore, there must have been doubts of the permanency of the fundal opening. Wertheim improved the technic by drawing the uterus through an anterior incision into the cul-de-sac; an anterior vaginal colpotomy instead of a posterior colpotomy, as done by Freund.[1] This method by Wertheim proved fully as efficient as that by Freund without the necessity for the fundal opening. The sutures were so placed that the body of the uterus could not escape from the vagina, and in turn the base of the bladder was elevated and sustained in a much better position. In passing, we call attention to the difference in the position of the uterus in these methods: the Freund uterus has its anterior wall sutured to the posterior vaginal wall, while the Wertheim method leaves the posterior wall of the uterus sutured to the anterior vaginal wall. One can see at a glance that both operations prevented impregnation and that the Freund method practically closed the vagina. At this time various suggestions were made which looked toward the substitution of an equally good if not better method of cure for the prolapse of both uterus and bladder which would avoid a resort to the objectionable use of the uterus as a vaginal plug to cure its own displacement. Accordingly, we find that operators in both Europe and America developed the plan of interposing the uterus between the anterior vaginal wall and the base of the bladder, which will always be associated with the names of Dührssen, Schauta and Watkins. The latter deserves the credit not only for early work upon this subject but also deserves the credit for popularizing the operation in the Americas. As

[1] See Döderlein and Krönig, also Am. Jour. Obst., May, 1918, p. 777.

all gynecologists agree, this operation is intended for such women with prolapse as may not or cannot bear children. Cesarean section has been performed several times upon women who have been subjected to this operation, and is a rational procedure if pregnancy should demand intervention in such a case. We are inclined to adopt the prevailing method of sterilization of women requiring an operation for prolapse prior to the menopause. As a rule they have borne several children and often appear to require every possible method of conservation of their health and strength. Sterilization is easily accomplished, as the fundus uteri is drawn with the tubes into full view and easy reach. They should be divided and one or both ends secured under a peritoneal cover.

The perfection of the operation requires a few words: From reports coming from many quarters one is to learn of possible errors to be avoided if one wishes to obtain uniformly good results. If the anatomical relations of the bladder are carefully studied, with a view to release a sufficient area at the base of the organ, there should be less fear of the convolutions of the mucosa which some authors have recently mentioned. We believe that a liberal separation of the bladder from the region near the cervix, and pressing the connective tissue widely right and left will serve to free the ureters and prevent danger of their kinking. (A fancy by some, but the writer has never known of such a case.[1])

The occasional use of the cardinal or uterosacral ligaments may be necessary in order to prevent the cervix from descending and appearing at the introitus. But for the unreliability of the uterosacrals we would have a satisfactory safeguard always at hand. However, if these ligaments are not competent, but few patients will suffer the least annoyance owing to this defect, because the restored perineum will usually prevent a return. We have spoken guardedly, for two

[1] See cut in Am. Jour. Obst., May, 1918, p. 777, from Sellheim.

possible conditions must be remembered: one of these is rapid foreshortening or cicatricial contraction of the anterior vaginal wall, the other is a deep cul-de-sac, which in certain instances comes nearly or quite to the levator ani muscle and must be considered a predisposing cause of prolapse. Such a condition is shown in a cut in the anatomical atlas by Sellheim. A section of a frozen pelvis clearly shows the explanation of some failures in operations for prolapse. The cul-de-sac is found very far down near the perineum, and the uterus is consequently without support from behind.

Results. The after-treatment of patients who have been subjected to this operation may have some influence upon the result. If the base of the bladder has been widely separated from its attachments, one may see instances of loss of function of the bladder for some days. This is never more than a passing annoyance which may necessitate the use of the catheter to prevent overdistention, and which in turn may result in a catheter cystitis. Intelligent use of the catheter and the prophylactic use of 2 per cent. protargol solution in the bladder will absolutely prevent subsequent cystitis. We find that 2 ounces of protargol solution used on two successive days will always prevent or relieve this annoying condition. The solution may be allowed to remain in the bladder until the patient's next urination.

Finally, the author wishes to call attention to the cure of involuntary urination by this operation. By reference to cut No. 5, p. 778,[1] in my article in the *American Journal of Obstetrics*, May, 1918, it will be shown how much nearer the pubis the fundus is placed than is usually seen in works on gynecology. The new and correct position for the fundus gives the urethra a curve somewhat like that of the male subject, which requires a curved or a soft catheter to reach the bladder. Fig. 5 is from a sketch made under the author's direction during an operation.

[1] Not cut No. 6, which was accidentally given the wrong description.

DISCUSSION

Dr. Franklin H. Martin, Chicago.—The operation that has been described by Dr. Stone is the most valuable procedure that has been brought to the profession in the last twenty-five years. It was popularized in this country by Dr. Watkins, and I believe that if it had not been for the work of Dr. Watkins, we in America would not have appreciated this operation. I believe, therefore, any discussion of this subject which does not give credit to Dr. T. J. Watkins should not be allowed to go by without calling attention to that fact. As you know, this operation for a while was called the Wertheim operation; afterward it was called the Wertheim-Watkins operation. It seemed to be a question as to who first performed the operation, Wertheim or Watkins. I happened to be in Vienna when Dr. Watkins and Dr. Wertheim met immediately after this controversy had been held, and it was curious to witness this meeting; but as it had already been recognized that the operation had been done eight or ten years before by, at least, two other operators, and they had already realized that neither of them could, while having derived the procedure, independently claim priority for the operation.

Dr. Charles R. Robins, Richmond, Virginia.—I have had a rather extensive experience with the operation described by Dr. Stone, having done it for a number of years. I had done the operation a number of times and had demonstrated how absolutely impossible it was to injure the bladder, when within a short time I had two cases in which I tore into the base of the bladder in pushing it up from the anterior surface of the uterus. There was no trouble, however, as a result of that; I simply sutured the bladder with a continuous chromic catgut suture and healing readily occurred. I know of nothing that heals more readily than the bladder; the blood supply is good and healing is prompt. When the Watkins operation is done a large number of times we will certainly injure the bladder in a percentage of cases. That is no detriment, however, because it can be so easily repaired and we never have any trouble from leakage.

Dr. Thomas S. Cullen, Baltimore, Maryland.—There is one point in the technic of the operation described by Dr. Stone

that I wish to draw attention to. After the bladder has been pushed up and the abdominal cavity opened the operator has been accustomed to grasp the anterior surface of the uterus with mesoforceps in order to bring the organ well within reach. The mesoforceps often tear out, leaving a rough and raw surface on the uterus.

During the last seven or eight years I have adopted a procedure that seems a distinct advance on the employment of instruments in bringing down and exposing the anterior surface of the uterus. When the peritoneal cavity has been opened and the bladder held up by a delicate retractor a small area of the anterior uterine wall comes into view. A figure-of-eight catgut suture, preferably chromicized, is then placed in the exposed anterior uterine wall and tied. Traction is now made on this ligature. The traction brings the uterus downward and forward for from half an inch to an inch. A second figure-of-eight is now introduced in a similar manner and tied. Traction is made on this second ligature, and a third figure-of-eight of chromicized catgut is introduced, tied and drawn down upon. Occasionally a fourth figure-of-eight suture is necessary, but as a rule by the time traction has been made on ligature number three the body of the uterus is sufficiently exposed.

After the cervix has been amputated or repaired the figure-of-eight sutures are picked up. The right half of each suture is then rethreaded, passed through the peritoneum and vaginal mucosa on the right, the left through the peritoneum and vaginal mucosa on the left. The bladder is then pushed up and the fixation ligatures tied in the usual manner.

Dr. Francis Reder, St. Louis, Missouri.—It is always of interest to have a rehearsal of so important and interesting a procedure as the interposition operation. The doctor has alluded to the technic where the flaps were cut too short. Originally it was the intention for the flaps not to meet in apposition but to allow a certain amount of space to intervene, which was to heal by granulation and cause a more firm fixation of the organ. I have tried both of these methods, and I must say that there is no difference, as far as the fixity of the uterus is concerned. There is a difference, however, in the length of time required for healing to take place. In the procedure where the flaps are made to appose, your patient will usually recover with a primary union in two weeks. Whereas, if a space for granu-

lation is allowed to remain it will be about seven weeks before the healing process has taken place. With reference to the ureters, I have paid but little attention to them in the uncomplicated cases. One complication that occasionally occurs in these cases is an obstinate cystitis, due to the extensive denudation of the bladder surface.

Dr. Guy Le Roy Hunner, Baltimore, Maryland.—I was very glad Dr. Stone emphasized the necessity of making a wide denudation of the bladder, and a thorough separation of the base of the bladder from the front of the uterus and from out on the sides. This certainly prevents a great deal of difficulty later in the operation when one wishes to bring the uterus forward and interpolate it. It gives another advantage which I have not heard mentioned, and that is the thorough exposure of the cardinal or broad ligaments, the basal ligaments. We find that these ligaments are hypertrophied in these cases and are sometimes as large as the little finger, and they ought to be made use of in this operation. Dudley, of Chicago, has done more, I think, to emphasize that point than anyone else, and it is not as thoroughly recognized as it should have been, namely, the great use of the broad ligaments in bringing them across the face of the cervix before covering the uterus with the vaginal flap.

Dr. Stone (closing).—I want to say in reply to Dr. Martin, that full credit is given to Dr. Watkins in my paper. I have looked over the literature very extensively, but could not cover it in the time allotted to me in the reading of the paper. Many things will appear in the paper when it is published that I did not have time to mention.

There are many points in connection with the technic about which we must more or less differ, especially in regard to the use of the broad ligaments, and that calls attention also to the uterosacral ligaments. Many rely on these ligaments for support, and I have tried myself to withdraw them and shorten them to hold the cervix very far back, but I would caution those who do that. In those cases where they are easily reached and handled, it is a simple matter, but there are instances when these ligaments are hard to pull out and to shorten successfully. But I have not had to use them save in a few instances. It is surprising how we learn to proceed from one step to another in the development of a perfect technic, and particularly so in the

operation under consideration. This operation is still being perfected; it is being developed, and there is undoubtedly a wide field of usefulness for it.

One word with regard to the urethra. In the cases where we have to deal with a decided leakage or inability to retain the urine, I have never had difficulty in absolutely relieving those patients of that trouble. In cases where there has been a leakage or insufficiency of the urethral muscle it is always necessary to elevate the urethra to quite a curve, with this operation. It should always be elevated to some extent, and the catheter is usually required on that account. It is a very important thing to elevate the bladder and the urethra with it, and consequently we have to caution the nurse in every instance, and sometimes instruct the intelligent intern, about catheterizing the patient.

Another thing which should be mentioned, and which has been referred to by Dr. Reder, is, the use of the catheter after these operations almost always causes a little cystitis. I invariably have some antiseptic agent at hand, say protargol, and use it at the conclusion of the catheter period whenever that occurs.

THE CAUSES AND SURGICAL ASPECT OF RUPTURE OF THE UTERUS DURING LABOR

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The sole object of this paper is to recall to your mind a subject with which some of you are quite familiar; but there are, no doubt, many present who have never been called upon to treat a case of rupture of the uterus during labor in which the cause was, strictly speaking, of an "obstetric" origin. The former will profit little as I review to them familiar scenes on a new screen; the latter may, perchance, be grateful to me for bringing back to their memories the sight of a catastrophe they once knew well, but which, because of its now rare occurrence, has become more or less effaced from their mental records. For aught we know the first case one may be called upon to treat on his arrival home may be one of those here considered.

Ordinarily, lacerations of the external os and of the vaginal portion of the cervix of the uterus are not spoken of as rupture of the uterus. Tears in this locality happen so frequently during labor and are of so little consequence at the time they occur that they often escape immediate observation.

Rupture of the uterus during labor, as ordinarily understood, is the result of prolonged and violent contractions of the uterus during the second stage of the act of birth, the period of expulsion. Rupture of the uterus during labor means a tear through the entire uterine musculature and,

not infrequently, through the parametrium and peritoneum. The rupture is called incomplete or non-perforating if the peritoneum is not included in the tear; if the peritoneum is involved, the rupture is said to be complete or perforating.

Rupture of the uterus is very rare. It is more frequent in multipara than in primipara. At the end of the first stage of labor, and throughout the period of expulsion, the uterus consists of two distinct segments: an upper or contractile portion representing the corpus uteri; and a lower or dilating portion made up of the entire cervix. The lower or dilating segment is more frequently involved in rupture of the uterus than the upper segment, and the tear is, almost always, the direct consequence of dystocia. The upper or contractile portion is seldom the site of a rupture and, when the accident does occur in this region, it is due to other causes than a prolonged and difficult second stage of labor. The rupture may be transverse, longitudinal, or oblique. Sometimes the entire uterus is torn from the vaginal vault.

The most frequent cause of rupture is a prolonged and difficult second stage of labor due to narrow and contracted pelvis, transverse presentations, faulty positions of the head and face, rigidity and displaced external os, and, it may be, the excessive size of the child, hydrocephalus, double monster, and tumors. When, on account of any of these conditions, expulsion is obstructed, continuous efforts on the part of the uterus result not only in a state of chronic contraction of the retracted corpus uteri, but also in an excessive overdistention of a very much attenuated cervix. In other words, the contractile portion of the uterus, unable to force the child into the pelvis, retracts above and draws the distended cervix over the body of the child. Thus the corpus uteri is more or less emptied of its contents at the expense of the overstretched cervix which now contains the greater portion of the child. The contraction ring—the ring of Bandl—becomes apparent or may be felt through the abdominal wall. This is the danger-signal preceding rupture of the uterus. The

element of traumatism is added if, under this condition, violent and prolonged attempts are made to deliver either with the aid of instruments or the introduction of the hand for the purpose of version.

Rupture occurring under the conditions just mentioned involves the lower uterine segment only, and the tear may be transverse, longitudinal, or oblique. The rent may take place in any part of the cervix: on the side near or in the region of the broad ligament; anteriorly, if the os is displaced posteriorly and upward, causing overdistention in the anterior lower uterine segment; or, when the cervix is acted upon equally from above and in every direction, the entire uterus may be torn from its cervicovaginal attachment.

Just when and where rupture of the uterus will take place, cannot be foretold. A normal cervical musculature, even when reduced to a few millimeters in thickness, may, under gradual dilatation, last a long time without rupture; while on the contrary, a pathological condition in this structure may predispose to rupture, and the accident may be sudden and unexpected. In the presence of excessive cervical attenuation when bulging occurs—as it sometimes does in a shoulder presentation or displaced rigid os—bundles of muscular fibers separate, and all that is needed to precipitate a rupture is the introduction of the hand for a version or the application of forceps. The child then passes through the slit in the muscular fibers and, if the peritoneum yields at the same time, into the abdominal cavity. If the serous membrane, which is but loosely attached in the region of the broad ligament, remains intact, only a portion of the child enters the parametrium; or, if the tear be not so extensive as to permit this, the blood of the torn vessels fills up the parametrium and a subserous pelvic hematoma results.

Cicatrices of a former incomplete tear, or an imperfectly healed uterine wound of a former Cesarean section or myomectomy, or an imperfect development or acquired atrophy of the uterine musculature may lead to early and unexpected

rupture of the womb. To the same category belong those cases in which the rent is the result of a kick, a bullet, a stab or the penetrating horn of a mad steer. As rupture of the uterus from these causes is exceedingly rare, atypical in character, and beyond anticipation and calculation, no blame can attach to the physician or midwife in attendance. Another form of rupture of the uterus is due to metal dilatation, particularly to the Bozzi and similar methods of dilating. Perforating ulcers of the uterus, the result of pressure necrosis, do not manifest themselves during labor and therefore should not be considered here.

When rupture of the uterus is not the result of an imperfectly united uterine wound caused by a Cesarean section, extirpation of a tube, a myomectomy, imperfect development or acquired atrophy of the uterine wall, etc., but is solely the consequence of an obstructed labor due to any one of the conditions stated above, its clinical picture is clear and definite.

He who has an opportunity to observe his patient closely from the beginning of labor will know whether the case before him will be a prolonged and difficult one. From the start he is on his guard if the case is one of narrow or contracted pelvis, a transverse presentation or some pathological condition of the uterine wall. The first stage of labor may pass without an unfavorable symptom. This will be followed by delay in the second stage. After the membranes have ruptured, there is either no descent of the presenting part of the child, or, if there is, such descent is very slow, and sooner or later is arrested altogether. The patient, at first restless, gradually becomes irritable and apprehensive. Later she refuses to be consoled, tosses restlessly from side to side, and seems powerless to assist in the expulsion of the child by using the auxiliary expulsive powers. The pains become severe, rending, and frequent in their recurrence. Examination shows a change in the shape and position of the uterus; it is very tender to the touch, especially so during a pain. The

fundus stands high up in the abdomen and is extremely hard. No part of the child can be felt through it. Through the lower uterine segment, soft, elastic and semifluctuant parts of the child may readily be recognized during the interval of pain. With advanced and extreme distention of the lower uterine segment there is marked retraction of the contractile portion of the uterus, and a line of depression runs obliquely or transversely, as the case may be, across the abdomen. This is the so-called ring of Bandl. It is the line of demarcation produced by the retracted corpus uteri above and the overdistended cervix below. It is commonly termed the contraction ring, and marks the location of the internal os. If the contraction ring runs obliquely across the abdomen, it is easy to determine the side upon which the cervical wall is subjected to the greatest pressure and where there is probability of rupture. Here, too, the round ligament may often be felt stretching like a rope over the distended cervix. The contractions of the uterus become increasingly painful; soon the pain becomes continuous, and the patient supports the lower abdomen with both hands in a mortal fear, plainly revealed in her face, that something is about to tear within her. As the gravity of the situation increases, the pulse becomes more frequent, the temperature begins to rise, and the pelvic soft parts become tender and dry. The climax has been reached. This is the clinical picture prior to rupture. It is a most impressive, indeed, a very striking one. No student of midwifery should engage in the practice of obstetrics until he has studied and mastered this array of symptoms in every detail. If no relief is afforded it is but the question of a short time when an agonizing, heartrending cry, followed by all the symptoms of shock and internal hemorrhage, will mark the moment when the overstretched tissues of the cervix yield to the enormous intra-uterine pressure. Rupture of the uterus has occurred. The catastrophe, in most cases, occurs suddenly and at the height of a pain.

If the fetus passes at once through the rent into the peritoneal cavity, the severe pain and feeling of tension which existed before disappear and all the symptoms of shock and hemorrhage manifest themselves. The pallid face portrays fright and great distress; drops of cold perspiration collect upon the forehead; nausea, vomiting, and syncope follow; the pulse is thready and very frequent. Inspection of the abdomen immediately after rupture may reveal the vigorous movements of the fetus; or if the child be dead, its body may readily be felt through the abdominal wall. The diagnosis of complete rupture is thus established. This picture, however, is often soon obscured by a rapid distention of the abdomen and great tenderness of its walls. Additional evidence of rupture may be obtained by digital examination. The part of the fetal ovoid, originally presenting, has disappeared. Another part has been substituted; or, if the child has slipped entirely into the peritoneal cavity, no trace of it may be felt on digital examination. Blood flows freely from the vagina; the examining finger may feel the tear and, in some cases, may come in contact with the slippery loops of intestines protruding through the rent.

If rupture of the uterus takes place more gradually, the symptoms are less violent because the fetus does not pass at once, if at all, into the peritoneal cavity. The peritoneum covering the broad ligament remains intact and is pushed up by the part of the child which the contracting uterus forces into the parametrium. Thus a vertex presentation may be changed into a shoulder, breech or footling presentation, and *vice versa.* In cases of this kind the symptoms of shock and hemorrhage may not be present, or may be manifested in no very marked degree. Occasionally the presentation remains unchanged; in that event there is no shock, but simply hemorrhage associated with a marked reduction of pain during uterine contraction. The continuous hemorrhage, a new and uninterrupted pain in the region of the tear, the formation of a hematoma in the same region,

together with a steadily weakening pulse and generally bad condition of the patient, help to make clear the diagnosis. Whether rupture of the uterus is sudden or gradual, whether the fetus be born into the peritoneal cavity or not, a complete or perforating tear of the womb is always a serious accident and one that is often fatal to both mother and child. The fetus dies before it is extracted; the mother is in danger of bleeding to death within a short time. If the hemorrhage can be controlled and the shock resisted a septic infection usually carries off the patient. Indeed, because of the protracted labor, frequent examinations and efforts of delivery, both manual and instrumental, the parturient tract may be already septic; if so, the rent in the uterus is quickly infected and a general fatal peritonitis is the inevitable result. The maternal mortality, collectively, of rupture of the uterus during labor is fully 33 per cent.

The Treatment before Rupture. The old adage that "An ounce of prevention is worth a pound of cure" has a particularly strong application here. One thoroughly versed in the science of obstetrics, who devotes his time and talent to his patient, will never have a rupture of the uterus from a purely obstetric cause such as narrow pelvis, faulty presentation, and position of the fetus, rigid or displaced os or a prolonged second stage of labor from any cause. He will know that with the appearance of the contraction ring the danger-line has been reached; but that the patient may still be considered safe so long as pulse and temperature remain normal and the genital canal continues moist and free from tenderness. If, in the case of narrow pelvis, there is evidence of configuration of the presenting head and progress in descent, waiting is still justifiable; but in the absence of progress, when labor is or has been at a stand-still, no time should be lost in effecting delivery. A careful version and the skilful use of the forceps are employed if the possibility of delivering by these means is apparent. In moderately contracted pelvis, between 8.5 cm. and 10 cm. in the antero-

posterior diameter of the inlet, if the child's life has not been compromised in the least, if the mother is free from sepsis, and the accoucheur familiar with hebosteotomy, this is the operation of choice in Germany; a Cesarean section would be the choice in this country and in England. If the conjugate diameter of the brim is less than 8.5 cm., the child living, the mother free from sepsis, and the environment favorable, Cesarean section should always be selected. If, however, the life of the child is already destroyed or seriously impaired, embryotomy in any form is the proper procedure; craniotomy, if the head presents; evisceration, in oblique presentation, etc. Great care must be taken not to disturb the uterus and pelvic soft parts when a version, the forceps, or the perforator is resorted to. Total anesthesia is necessary for the successful execution of these operations.

The Treatment after Rupture. If the rupture of the uterus is incomplete the child should be delivered through the parturient canal if this method be possible; in case of vertex presentation the forceps must be employed with or without perforation of the head; or delivery may be accomplished by traction upon the feet, if the breech or the feet present; perforation of the after-coming head is sometimes necessary. If rupture is complete, the child may still be extracted *per vias naturales* if the greater part of its body still rests within the uterine cavity. Whether the tear is complete or incomplete, if delivery has been affected through the natural channel, hemorrhage may be successfully arrested by tamponing the uterine cavity and the vagina and by forcing the uterus into the pelvic cavity by placing a firm compress upon the abdomen. In some instances hemorrhage ceases spontaneously, and an ice-bag upon the abdomen, a hypodermic of morphin and complete rest in bed, have been all that were needed. The peritoneal rent, which closes promptly within twenty-four to forty-eight hours, removes the greatest danger.

If the child escapes into the abdominal cavity a celiotomy

alone can save the patient. After the child and placenta have been removed the peritoneum is cleaned, the rent in the uterus is sutured and the serous membrane is carefully sewed over the latter. If sepsis exists in the case the uterus and appendages must be removed.

The history of rupture of the uterus during labor strikingly illustrates the importance of conveying to a hospital women in or approaching a complicated labor. In private practice, unless asepsis has been maintained throughout and a competent operator has been in charge of the case, delivery *per vaginam* will be the method adopted whenever possible; in hospitals where all is ready for an operation, celiotomy will be the operation preferred, because it gives the mother the best chance of life. We should always remember, however, that in the presence of sepsis, no matter what the surroundings and how skilful the operator, death always follows, while under aseptic conditions women have made excellent recoveries after the extraction of the child through the vagina.

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CESAREAN SECTION UNDER LOCAL ANESTHESIA

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In presenting this paper we appreciate fully we have employed local anesthesia in some cases in which there were no contra-indications to a general anesthetic. However, as all such patients have had so little pain and no shock, we feel justified in having followed this course in order to test the practicability of a method to be applied in those cases in which a general anesthetic adds gravity to an already grave situation.

Since Einhorn gave to the surgical world novocain, most of the major operations of surgery have been performed with its use, but a search of literature failed to show an abdominal Cesarean section done with this anesthetic up to the time of our first case. However, since then, Dr. Clarence Webster reported 14 cases in *Surgery, Gynecology and Obstetrics,* February, 1915—most of his cases preceded our work along this line, and he certainly deserves all credit for priority.

In our series we have had 18 Cesarean sections done with local anesthesia, and a considerable number of others with general.

Why this field of surgery has not been more utilized by the advocates of local anesthesia is difficult to understand, for frequently where the necessity for such an operation is present the contra-indication to a general anesthetic is marked, and we are convinced the procedure offers few real difficulties, and can be done with relatively little pain—certainly nothing like as much as normal labor.

It is, of course, necessary for any surgeon attempting this

work to be accustomed to the employment of local anesthetics, and any operator expecting to gain entrance painlessly into the abdominal cavity, should certainly be familiar with the great work of Lennander on the sensibility of the various viscera and parietal peritoneum. Curious to note, Lennander does not mention the sensibility of the uterus either in the pregnant or the non-pregnant condition, further than to state that "All organs receiving their nerve supply only from the sympathetic nerve and from the vagus, below the branching off of the recurrent nerve have no sensation." "According to my observations, therefore, the abdominal and pelvic viscera are devoid of nerves to convey the sense of pain, pressure, heat or cold."

The truth of the above quotation has been impressed upon us very forcibly during our operations on the pregnant uterus, namely, the patients have no pain when the uterus is being incised, but do complain of nausea and pain to much traction, which latter condition is what would be expected, but certainly the former state of affairs is an important and somewhat unexpected observation. We have purposely told four patients before we started to incise the uterus, and none have complained of pain during the entire incision, but all did have pain when we lifted the child and membranes from within the uterus. This, of course, helps prove Lennander's contention that there is pain to the slightest traction on the mesentery or parietal peritoneum. This point is further demonstrated in the fact that in all 18 of our cases the most painful part of the operation was the lifting of the uterus out of the abdominal cavity.

The technic of the operation, as we have followed it, is briefly as follows: Operation preceded by morphin $\frac{1}{6}$, novocain 0.5 per cent. with 3 drops of adrenalin to 1 ounce is the solution employed, and of this we have used as high as 250 c.c. In the last 11 cases we have omitted the adrenalin and could see no difference in the duration of the effect of the anesthetic or the amount of bleeding.

Skin infiltrated in the usual manner by forming one wheal after another. In the first 5 cases we made the incision from the pubes upward until we could have room to deliver the uterus, but during the last 13 we made the middle of the incision about the level of the umbilicus, and the upper end about on a level with fundus of pregnant uterus (Fig. 1). The fascia is infiltrated exactly in the same manner as the skin, and muscles are usually so thin the fibers separate without trouble. A small opening is then made in the peritoneum, and index finger of left hand inserted and peritoneum infiltrated, keeping this finger on the inside as a guide for the needle while injecting this very thin membrane. This part of the operation is far easier than is commonly supposed. The uterus is lifted out of the abdominal cavity, and moist sodium citrate and sodium chloride pads or towels placed between it and the intestines.

It is important to have the upper portion of the abdominal incision so placed as to be slightly higher than the fundus of the pregnant uterus, and in this manner the uterus can be allowed to "ride out" of the peritoneal cavity; thus removing what is sometimes the only painful part of the operation. If the incision is not too long, the abdominal wall will "hug" the uterus and serve to retain the intestines without the use of pads, etc. (Fig. 2).

As a matter of precaution, and to save time after incision of the uterus, we place a line of interlocking sutures of chromic catgut on each side of the fundus of the uterus (Fig. 3). These lines being about one inch apart. The sutures are placed as follows: The points of entrance and exit are about one and a half inches apart. The next suture has its point of entrance half-way between the two above points, and this manner of inserting sutures is continued until about eight to ten sutures are placed in each side of the future line of incision. As these sutures are placed in the direction of the long axis of the uterus, it is easily seen a line of sutures so placed will interlock one another and control

all bleeding. These sutures go through the whole muscular wall, and an assistant on each side makes traction on the entire line, thus lifting the uterus up, steadying the same, and controlling the hemorrhage (Fig. 4). After delivery of the child and membranes, these sutures are tied across the line of incision, and in this manner we have at least eight double mattress sutures which not only control the hemorrhage but give fairly accurate approximation (Fig. 5). This whole suture line is covered over by means of a continuous suture of plain catgut, approximating the peritoneal surfaces, leaving no raw surfaces for future adhesions (Fig 6). The abdominal incision is closed in the usual manner.

The following is a very brief synopsis of the 18 cases:

Nine cases of eclampsia. Three mothers died, all having had convulsions before operation; six children born dead, three living. Very little dilatation of cervix in any of these cases. Forceps delivery attempted previous to operation in all 3 cases that died; no forceps in the remainder of the series. All cases having had convulsions presented an aura of radiating epigastric pain. There were six primiparæ and three multiparæ.

Five cases of contracted pelvis. All mothers living, three children living and two born dead.

Two cases of placenta previa. Both mothers living.

One case of pyelonephritis; not relieved by ureteral catheterization, etc., and patient very toxic. Mother living, child born dead.

One case of uterine inertia in primipara. In labor over forty-eight hours without any dilatation and collapse of mother. Mother and baby living.

From our experience with those cases we are convinced where the condition of the mother makes the giving of a general anesthetic unsafe the employment of novocain is indicated and its use does not present the difficulties one would naturally expect.

Fig. 1

Fig. 2

Fig 3

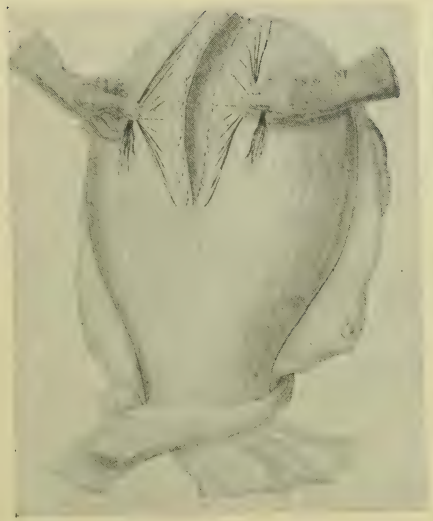

Fig. 4

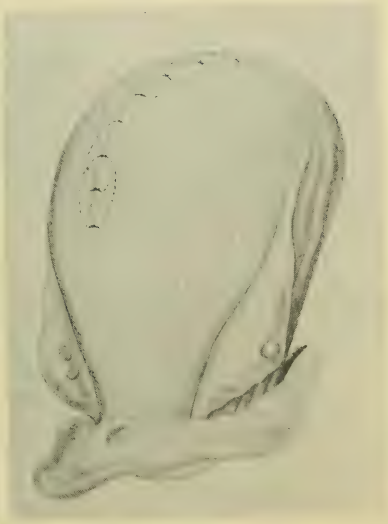

Fig 5

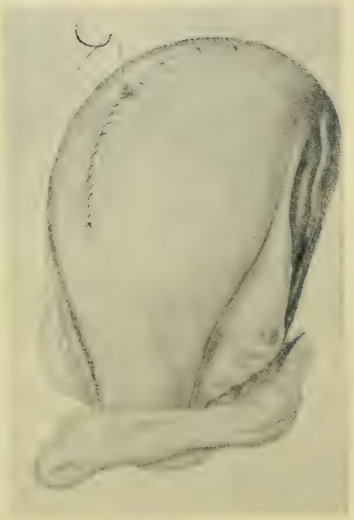

Fig. 6

DISCUSSION ON THE PAPERS OF DRS. ZINKE AND TROUT

Dr. I. S. Stone, Washington, D. C.—I do not know that I can fully appreciate Dr. Zinke's calmness of mind when he described his patient as having rupture of the uterus, and the question arises whether delivery should be effected *per vias naturales*, by forceps, or by some other method. Is the hemorrhage persisting or has it ceased? If it has ceased, we would sometimes hesitate to perform an operation through the lower route. On the other hand, if in our opinion the uterus is ruptured above, at or near the fundus, it seems to me the tendency at the present time would be directly in favor of Cesarean section.

I have enjoyed Dr. Zinke's paper very much, but having decided that a rupture of the uterus has occurred and that hemorrhage is persisting, or likely to persist, I do not see how we can hesitate, surgically speaking, to go right after the source of rupture and hemorrhage. I think, without any doubt, we would in all instances go after the hemorrhage at its origin.

Dr. F. W. Parham, New Orleans, Louisiana.—I was very much interested in Dr. Trout's paper on the question of local anesthesia, because many of us in New Orleans have found the field is materially increasing in which we employ local anesthesia.

I am glad to hear that the doctor was so successful in his 18 operations, but was surprised to hear him say in the last 3 cases he had found the absence of adrenalin of no consequence; that the anesthesia was as good without as with the adrenalin. That may be explained by the fact that Cesarean section in its performance is a comparatively short operation, so that the adrenalin effect did not pass off. Braun in his book on local anesthesia emphasizes the fact that the addition of adrenalin enables the surgeon to substitute novocain for cocain, which is the more dangerous anesthetic.

We have had some trouble in getting novocain in New Orleans, and we have been lately employing apothesin with satisfactory results. It is less toxic in its effect than even novocain and is absolutely as anesthetic, so far as our observation and experience go. I have not seen any untoward effects from the use of apothesin.

Dr. John Wesley Long, Greensboro, North Carolina.—Several years ago, through the courtesy of a distinguished Founder of this Association, Dr. I. S. Stone, I was invited to read a paper before the Washington Obstetrical and Surgical Society. The subject of my paper on that occasion was "Rupture of the Uterus." In the preparation of the paper I made a fairly careful search of the literature of the subject. To give you some idea of the amount of work involved, I may say that I reviewed a million cases of labor, taking the statistics from the New York Lying-in Hospital, the Berlin hospitals, the Moscow hospitals, Rome, Vienna, and others.

There were several things in connection with the subject of rupture of the uterus that were very surprising to me. Perhaps the most striking observation was that in many of these cases the rupture began from the peritoneal side instead of from the mucosa of the uterus. This was determined by both operative and autopsy findings. In addition to the complete through-and-through ruptures many partial ruptures extending through the peritoneum and partially through the muscularis were found.

In this paper I reported the only case of rupture of the uterus, barring perforation of the uterus from instrumentation, that has ever come into my hands. The patient was a rather old primipara, who was under the care of some very competent general practitioners. The head was bulging from the perineum. Following a severe pain the head disappeared. On vaginal examination the accoucheur's hand passed through what proved to be a rent in the uterine wall. The baby had escaped into the abdominal cavity. Catching the feet the delivery was easily accomplished.

I saw the patient about four hours after the delivery. She was removed sixteen miles to the hospital, and twelve hours after the accident I opened the abdomen and did a supravaginal hysterectomy.

A noticeable feature was that the rupture which was complete was situated entirely below Braun's ring and extended far out into the broad ligament. I have a lantern slide of the specimen, and I am sorry I did not bring it along, because it shows Braun's ring very strikingly and as thick as the base of one's thumb. This rupture took place in front of the uterine artery, so that there was very little hemorrhage. The woman made an uneventful recovery, having no rise of temperature for a week. At the end of that time she developed a classical secondary anemia, as

was shown by the blood findings. However, she successfully passed through the secondary anemia, which was by far the most serious part of the whole affair so far as the woman was concerned.

Dr. J. M. Mason, Birmingham, Alabama.—I have enjoyed both of these papers. The statistics given by Dr. Trout are particularly interesting. While it is a matter of discussion whether puerperal eclampsia in a patient who has not yet gone into labor should be treated by expectant methods or by rapid delivery, I am firmly of the opinion that rapid delivery is the proper procedure and that this can be best accomplished by Cesarean section unless the cervix is short, soft and easily dilated, and unless the pelvis and vagina are very roomy. The great danger has been not so much the operation, which is not as shocking, in many instances, as prolonged labor, but the effect of the superadded general anesthetic which has heretofore been used in the Cesarean section. In these cases, where the woman comes into the clinic either in convulsions or in coma, or seriously threatened with convulsions, and where the cervix is tightly closed, as is often the case, and where labor has not begun, and you cannot, with safety, hurry up things from below, the use of local anesthesia, as presented in these 18 cases by Dr. Trout, removed a serious drawback to Cesarean section. With local anesthesia and rapid Cesarean section many lives will be saved in the future which would be lost if treated otherwise.

Concerning Dr. Zinke's paper I wish to say: He states that when infection is added to rupture of the uterus, the termination is invariably fatal. I wish to report one case of that character which did not end fatally.

Three years ago a colored woman, primipara, about thirty-eight years of age, was brought into the Hillman Hospital after having been attended by a midwife for thirty-six hours without effecting delivery. She was delivered by forceps by the visiting obstetrician, with great difficulty. A rent occurred in the left vaginal fornix, extending well up to the cervix and out into the broad ligament, and, we thought, up into the peritoneum. She had a temperature of 102° and a foul discharge. Two hours after the delivery, I did an abdominal hysterectomy with drainage, and she made an uneventful recovery. The rent, however, had not extended to the peritoneum, but did extend high in the broad ligament on the left side.

Dr. Herbert P. Cole, Mobile, Alabama.—I would like to say a few words in reference to Dr. Trout's paper on local anesthesia in cases of Cesarean section. I think many of us overlook a great many points in doing laparotomies under local anesthesia, especially the use of a preliminary narcotic. Many men, who use local anesthesia, find that a stable solution of scopolamine, given in two doses several hours apart, will widen the adaptability of local anesthesia, especially in gynecological cases.

There are one or two points in the administration of local anesthesia that appeal to me and to others who have used it successfully. One is to use enough of the local anesthetic; second, to give enough time for it to produce its effects; third, to be sure and thorough in the administration of the anesthetic, to give it intradermically, and not open the tissues until the infiltration is complete. It is necessary for one to revise his method of operating under local anesthesia. One should bear in mind the making of large incisions to avoid unnecessary traction of the edges of the wound; the non-use of retractors, extreme gentleness in handling the viscera, the intestines and omentum, being careful to separate all adhesions with sharp dissection. You cannot do operations very successfully under local anesthesia with blunt dissection. It is vivisection and not local anesthesia. There should be the careful use of warm solutions, avoidance of the bowel and viscera by moist gauze gently placed. If traction is used, it should be made with the gloved hand. If it is necessary to force the viscera to one side or to expose them, it can be done with a flexible spatula which prevents the patient from being shocked. The uterus can always be freely exposed through a large incision with the patient in the Trendelenburg position. The absolute absence of sensory pain when an operation is done on the uterus is manifest during the operation.

Some years ago, at Cincinnati, I reported several hundred cases of laparotomy under local anesthesia. One patient was an eighteen-year-old girl from whom I removed a forty-pound ovarian cyst under local anesthesia. Since that time I have performed hysterectomies under local anesthesia, and this summer I removed a fourteen-pound myoma from a woman, aged sixty years, with a bad cardiac lesion, under local anesthesia without particular discomfort.

After Cesarean section under local anesthesia there is no

difficulty in closing the abdomen, as the stretched abdominal wall coapts after removal of the child. Dr. Trout spoke of using 20 grains of novocain. I have used 26 grains in operating for an umbilical hernia. Adrenalin is administered to diminish the amount of anesthetic used. Local contraction of the blood-vessels prevents more or less absorption of the novocain.

I have had such gratifying results from operating under local anesthesia that I only resort to general anesthesia now in cases in which I think it is absolutely necessary.

Dr. Charles R. Robins, Richmond, Virginia.—In reference to rupture of the uterus, one source of hemorrhage is in the venous plexuses. We are all familiar with the fact that at the end of labor the veins are enormously distended and extremly tortuous, and I believe that in the vast majority of cases, where we have a fatal hemorrhage from rupture of the uterus, it is from the venous plexuses and not from the uterine artery. If we have an injury to the venous plexus, the vein being as large as the little finger, the bleeding is continuous, and there is no attempt at spontaneous stoppage. I believe that is one of the reasons why we should always err toward opening the abdomen in preference to trying to complete the operation from below.

Dr. E. Gustav Zinke (closing the discussion on his part).—It is not quite clear to my mind why we should resort to local anesthesia to perform Cesarean section when the other anesthetics, such as chloroform, ether, or both, or the A. C. E. mixture have been so satisfactory. General anesthesia saves the woman the shock that she must necessarily experience when she witnesses this formidable operation upon herself. I am not favorably impressed with the performing of Cesarean section under local anesthesia.

Pardon me for referring to surgery for the relief of eclampsia. I discussed this question at the meeting in Washington. There is really no place for surgery in the treatment of puerperal convulsions, for it has not added one iota to the reduction of the maternal mortality of this disease. Cesarean section, which I advocated for placenta previa more than fifteen years ago, is now recognized as the proper procedure in these cases.

In reference to Dr. Stone's remarks as to what should be done prior and subsequent to rupture of the uterus, I have addressed myself to both the abdominal specialist as well as the

general practitioner. A well-trained and conscientious obstetrician may be called upon to treat, in private practice, a case of threatened or of actual rupture of the uterus during labor, and not be able to send the patient to a hospital for the purpose of performing an abdominal section. To perform a laparotomy at home would, probably, be fatal, because of what has already taken place, and because of the patient's usually unfavorable environment. The situation is entirely different if the patient has been in the care of a competent obstetrician, and in a well-equipped hospital from the start. Under such circumstances, when rupture has actually occurred, an immediate abdominal section should give the best results for both mother and child. There are cases of incomplete rupture of the uterus on record in which the child has been successfully extracted *per vias naturales*, and the mother recovered. In these cases, however, the hemorrhage is not, as a rule, very excessive, and coagulation of the blood soon takes place. The reason for the small loss of blood is, probably, explained by Dr. Robins, and shows the justification for delivery *per vias naturales* in these cases.

None of us have had extensive experience with cases of rupture of the uterus. I have seen only three of them in forty-three years of practice. All of these cases occurred in the care of other men.

Dr. Trout (closing).—We found ourselves in the position that Dr. Parham has described, and not being able to obtain novocain we tried also apothesin, which was sent to us by Parke, Davis & Company. We tried it in two cases and found it satisfactory, and when finding we could not obtain any more, we tried another preparation called alypin, which is used by ear, eye, nose and throat men. I know very little about it, but it was not satisfactory for abdominal work.

In reply to Dr. Zinke, I wish to state we have used genera anesthesia in cases of eclampsia and the patients have not done as well as they have done with local anesthesia, and this also has been the experience of a number of my surgical friends.

A comparative study of the mortality of a series of cases of eclampsia given general anesthesia with a series done under local anesthesia shows a far lower mortality in favor of the local, both as regards mother and child.

THE TREATMENT OF CERTAIN FORMS OF SUBACUTE PANCREATITIS

By Stephen H. Watts, M.D.
Charlottesville, Virginia

I believe all surgeons are agreed that those cases of subacute pancreatitis in which there is abscess formation should be operated upon and drained, the abscess often being indicated by a palpable mass in the pancreatic region, sometimes filling the lesser peritoneal cavity and even bulging the abdominal wall; but I believe little attention has been directed to those cases of subacute pancreatitis in which the symptoms at time of operation are not very acute, a diagnosis of gall-stones is made and at operation the pancreas is found to be thickened and indurated but still fairly well defined.

In these cases there is little or no free fluid in the abdominal cavity, and this not blood-stained, attention being attracted to the pancreas only by the finding of disseminated fat necroses in the omental fat or in the peripancreatic tissues.

In previous papers I have emphasized the importance of early incision and drainage of the pancreas itself in cases of acute pancreatitis and have reported 4 such cases, in 3 of which recovery followed drainage of the pancreas; in the remaining case which was a fulminant one, the pancreas was not drained and the patient died; however, I believe there would have been a fatal result in this case under any conditions. In the light of certain cases which I will presently report I believe that the same thing should be done on those cases of subacute pancreatitis which seem to verge almost

on the chronic; in fact, I am inclined to believe that some of these represent an acute exacerbation of a chronic pancreatitis. I have operated upon 3 cases which seem to belong in this category. In all of these cases a diagnosis of gall-stones was made, gall-stones were found in the gall-bladder and a cholecystostomy was done but the pancreas and peri-pancreatic tissues were not drained. In all of the cases fat necroses were found in the omental fat or in the fat about the pancreas, and the pancreas itself was somewhat, in fact, in 1 case markedly, thickened and indurated, but the outline of the pancreas was fairly well defined. The condition, except for the fat necroses, suggested a chronic pancreatitis; therefore the organ was not drained. One of these cases recovered and was in good condition when seen a year after the operation. The other two died, the one nine, the other sixteen days after the operation. Autopsies revealed extensive necroses of the head of the pancreas in both cases and in one there was extensive hemorrhage in the tissues about the pancreas.

I believe that if pancreatic drainage had been instituted in these cases the result might have been different. The case reports follow:

Case I.—Mrs. W. M., aged thirty-six years, was admitted to the hospital April 14, 1911, complaining of pain in the abdomen radiating to the back.

Family History. Unimportant.

Personal History. Has had measles, mumps and whooping-cough. Had typhoid fever ten years ago. Has suffered with stomach trouble for eight years, manifested by vomiting and pain in the stomach radiating to the back. Has had nine children, youngest being one month old.

Present Illness. Patient gave birth to a child four weeks ago. Before the birth of the child she suffered from burning, cramp-like pains in the stomach, passing to the right shoulder, and these pains have become worse and continued to the present time. They have required frequent hypodermics

of morphin. Six days ago she vomited a large amount of bile-stained material. At that time she was jaundiced. She had chills every night for a week after the birth of the child.

Examination. Patient is a thin woman, looks sick. Sclerotics are distinctly bile-stained. Pulse is of fair quality, 92 to the minute. Temperature, 100° on admission. Leukocytes, 9200. Coagulation time, four minutes. Urine analysis negative. The abdomen is flat and slightly asymmetrical, there being a fulness in the right hypochondrium. On palpation there is considerable tenderness in the epigastrium and right hypochondrium. No mass can be felt in the epigastrium, but in the right upper quadrant the edge of the liver can be felt 5 cm. below the costal margin, and beneath this a rounded body, the size of a hen's egg, which feels like a distended gall-bladder. On inspiration it descends almost to the level of the umbilicus.

Operation. Incision was made through the upper portion of the right rectus muscle. The gall-bladder was found to be considerably distended and numerous stones could be felt in it. The gall-bladder was aspirated and a large amount of bile obtained, which would seem to indicate an obstruction of the common duct rather than of the cystic duct. The gall-bladder was then opened and numerous small, faceted stones and one stone the size of a walnut removed. A careful examination of the common duct failed to reveal any stone. The pancreas was examined and the whole organ found to be greatly enlarged and fairly fixed in position. In places the limits of the gland were not well defined. In the transverse mesocolon near the pancreas there were several areas of fat necroses, suggesting a subacute pancreatitis.

A large rubber tube was sutured in the gall-bladder and the abdominal wound partly closed.

Postoperative Course. The patient did fairly well for a few days after the operation, then began to run a temperature and the pulse became more rapid. She died rather suddenly nine days after the operation.

Autopsy. About a liter of blood was found in the abdominal cavity. Mesentery of the ascending colon infiltrated with blood. Head of the pancreas converted into necrotic, hemorrhagic mass. Numerous fat necroses found. The pancreatic ducts were patent but a firm calculus 5 mm. in diameter was found in the ampulla of Vater.

Case II.—Mrs. J. A. H., aged forty-eight years, entered the hospital February 17, 1916, complaining of stomach trouble.

Family History. Unimportant.

Personal History. Has never had typhoid fever. Had pneumonia nineteen years ago. Has suffered with "indigestion" for years. Has had four children, no complications during labor or puerperiums.

Present Illness. About a year ago she had a severe attack of epigastric cramp, mainly upon the *left* side. Pain was intense, lasted about six hours. Since then she has had at irregular intervals similar attacks, sometimes with vomiting, sometimes without. There has been little if any temperature. Preponderance of pain has been in the left epigastrium, some, however, in the upper right abdomen with occasional radiation to the back. She has never vomited blood. Her last attack, which was very severe, occurred the day before admission and lasted about six hours.

Examination. Patient is moderately nourished, appears rather weak. Sclerotics distinctly jaundiced. Temperature, 100°; pulse, 100, of fair volume and tension. Urine examination shows a trace of albumin, no sugar. Abdomen is flat. No visible masses. There is tenderness across the whole upper abdomen, especially in the midepigastrium and a few inches to the left. No masses felt. Liver dulness reaches the costal margin in the right nipple line. A diagnosis of gall-stones was made.

Operation. April 20, 1916. A high right rectus incision. On opening the peritoneal cavity no free fluid was noted. The gall-bladder was found to be somewhat thickened and

to contain gall-stones. Numerous fat necroses were found in the omentum and peritoneal fat. The pancreas was markedly thickened and firm throughout. No stones could be made out in the common duct. One large and several small, mulberry stones were removed from the gall-bladder and cholecystostomy done. The pancreas was not drained. This seems to be an example of an acute exacerbation of a chronic pancreatitis.

Postoperative Course. Patient had an uneventful convalescence. Wound healed nicely. General condition good at time of discharge, four weeks after operation.

Case III.—Mrs. J. H. H., aged forty-eight years, was admitted to the hospital April 8, 1916, complaining of pain in the stomach.

Family History. Unimportant.

Personal History. Patient has been having attacks similar to the present one for the last five or six years, coming on at intervals of several months and often lasting for several days, but none has been as severe as this one. No history of previous jaundice, chills, or fever. Bowels somewhat constipated and stools of yellow color. No urinary disturbance. She is the mother of eight children.

Present Illness. About ten days before admission patient was seized with severe cramp-like pain in the epigastrium, radiating to the back, more to the *left* and also down into the lower abdomen. She suffered much pain and vomited almost continually for a number of days. Pain has been almost constantly present since the onset. Says she has been very tender in the upper abdomen.

Examination. Patient is a rather stout woman. General condition seems fairly good. Skin is slightly yellow and sclerotics are definitely bile-stained. Temperature, 101°; pulse, 90, of good quality; leukocytes 15,000. Urinalysis negative. Abdomen is not distended. There is considerable tenderness in the gall-bladder region and as far down as the appendix region. There is some tenderness in the epigas-

trium and to the left. No masses felt. Liver and gall-bladder not felt. No dulness.

Operation. April 10, 1916. High right rectus incision. No free fluid in the peritoneal cavity. The omentum was found adherent to the gall-bladder and showed numerous patches of fat necroses. The gall-bladder was moderately distended and contained one large stone without facets. There was considerable induration around the common duct but no stones could be felt in it. The pancreas was much thickened and indurated throughout its extent. A rubber tube was sutured into the gall-bladder and iodoform gauze packed around the gall-bladder and toward the foramen of Winslow.

Postoperative Course. Patient did fairly well for four or five days after operation. She then became more deeply jaundiced, began vomiting. Wound broke down throughout and discharged bile and a thin, brownish fluid. Vomiting became persistent. Pulse became rapid and weak. Temperature, which had been elevated since admission, gradually rose, and during the last days ranged between 104° and 106°. She died sixteen days after the operation.

Autopsy. A postmortem examination was made and revealed a subdiaphragmatic abscess, extensive fat necroses in the great omentum, mesentery, subperitoneal fat and about the liver and right kidney. The pancreas was adherent and was dissected out with difficulty. Its head was entirely necrotic, leaving nothing but a blackish débris. Its body and tail were intact, but quite hard and showed nothing unusual on section. Pancreatic duct necrotic and could not be found. No obstruction found in the common duct.

DISCUSSION

Dr. F. G. Du Bose, Selma, Alabama.—I would like to contribute one case to the interesting paper of Dr. Watts. A negro woman was sent into the Colored Infirmary with a diagnosis made by a physician of fibroid tumor of enormous size. An assistant was sent to the infirmary to make an examination and the usual routine tests, blood tests, urine tests and so forth, and to prepare the woman in case she was found safe for operation. He reported that her pulse was rapid; that otherwise conditions were normal and she had an enormous fibroid. The abdomen was greatly distended. I did not myself make a preliminary examination nor an examination of the feces. On opening the abdomen I saw this enormous tumor with the stomach and duodenum spread out over it and adherent densely and closely to it. On dissecting up the colon, as soon as I lifted the colon and omentum and dissected the colon loose from this, I recognized she had an enormous retroperitoneal tumor the character of which I had never seen before. We proceeded with our dissection, and when we finally loosened the stomach and duodenum, we recognized the fact that we had a pancreatic tumor. It included practically all of the pancreas except the distal three and a half or four inches. This was amputated, the tumor removed, and the distal end of the pancreas stuck into the duodenum with a double row of sutures. As soon as we saw the magnitude of the tumor she had, we resorted to the use of hypodermoclysis, and intravenous transfusion. The tumor, after its removal, was examined and found to be a sarcoma of the pancreas. It was globular in form, and measured about fifteen inches in diameter. This woman lived only seven or eight hours, and died as a result of the operation. So far as the treatment of pancreatitis is concerned, I agree with the essayist that drainage of the gall-bladder at present is our best means of treatment.

Dr. Alexius McGlannan, Baltimore, Maryland.—We owe our thanks to Dr. Watts for calling our attention to this method of dealing with a serious complication of chronic cholecystitis, with jaundice. If any member of the Association has had the experience which Dr. Watts has related of the sudden death of a patient several days after operation with signs of

severe infection, should a postmortem examination be made, he has found the difficulty in the pancreas, and not in the liver, where we ordinarily expect the infection to show itself in such a disease. If we will do as Dr. Watts recommends and, in addition to draining the gall-bladder, make direct drainage of the pancreas, we will find that we can avoid such a disaster. Should the pancreas go on to further dissolution and infection, we will provide an outlet for the toxic material which forms, and we give the patient an opportunity to get well rather than to absorb the toxic material and die with what is practically, as far as the end-result goes, a fulminating acute pancreatitis.

Dr. Lewis C. Morris, Birmingham, Alabama.—I believe that there is a difference between a subacute pancreatitis associated with infections of the gall passages and chronic pancreatitis secondary to acute pancreatitis.

About fifteen years ago I operated on a case in which there was marked enlargement of the head of the pancreas associated with stones in the gall-bladder. In this case the induration and enlargement were so great that I thought it was definitely a case of malignancy of the pancreas. I removed the stones and made an effort to get a specimen from the head of the pancreas for pathological examination. In attempting to get the specimen a rent was made in the portal vein which I had to clamp and tie off; and no further effort was made to secure the specimen for pathological examination. This woman made a good hospital recovery, and went home with the prognosis that she would probably die within a year or more. This was about fifteen years ago. Two years ago I was in the same section she came from and had the pleasure of taking lunch at her house. This patient had a chronic pancreatitis, and not malignancy, which was secondary to an inflammatory condition of the gall-bladder and liver ducts.

Dr. George A. Hendon, Louisville, Kentucky.—I would like to ask Dr. Watts, in closing the discussion, to state whether or not in the cases he operated upon there existed pancreatitis of whatever grade—subacute, acute or chronic—with an associated cholecystitis, or if there were gall-stones present. Also, if there is any method or means by which we are able to recognize chronic or subacute pancreatitis before operation is done.

I am particularly interested in obtaining some information

regarding the associated conditions. I rather feel that in all cases of pancreatitis we have an associated cholecystitis, and *vice versa.*

Dr. Joseph Ransohoff, Cincinnati, Ohio.—I will be glad to relate a case of chronic or subacute pancreatitis associated with cholecystitis. I am glad that statement was made because I do not think I am sure that cases of acute cholecystitis are the result of trauma, but I told Dr. Morris that if he ever had an opportunity of opening my abdomen and found something wrong with the pancreas, not to cut out a piece of tissue unless it was absolutely necessary; if there is any good way of producing an acute pancreatitis, which is not of necessity of parasitic origin at all, it is by producing a wound of the pancreas. The smallest kind of wound of the pancreas is apt, by reason of autodigestion, to do an enormous amount of damage, and for that reason it is not good surgery to cut out a piece of the pancreas unless you have a tumor that you know is limited and are getting only a piece of the tumor tissue. To take out a portion of the normal pancreas for pathological examination, I think would be bad surgery.

I would like to ask the essayist, in closing the discussion, to tell us how often he has made a diagnosis of acute pancreatitis. I have seen not a few of these cases, and I do not recall one in which the diagnosis was made. I feel sure that everyone who has had cases of acute pancreatitis is able to make the diagnosis when the abdomen is opened, with a large quantity of blood in the abdomen, and find evidences characteristic of the disease. However, I know of no way of making a diagnosis of acute pancreatitis, and I do not know that anyone else does. If the acute case has gone on to the degree of suppuration or pus formation, if the case is not one of acute hyperpancreatitis, it is possible to make the diagnosis.

With regard to making a diagnosis of pancreatitis that is not acute, that is subacute or chronic, diagnoses are made not infrequently, and upon what? Not upon the clinical history, but on what is found at the operating table, and what is found at the operating table? Is the pancreas ever exposed as it is in an autopsy? No, I do not think it is. We are guided largely by what we feel, and the diagnosis is not infrequently made aside from the clinical history. We base our diagnosis upon the increased hardness of the pancreas, and upon the slight increase

in size of the head of the pancreas. Those are the two main things upon which the diagnosis is made. I would suggest, in order to make this diagnosis, to go into the postmortem room after the subject is opened and the pancreas is exposed, and see the enormous variation in the sense of resistance to the finger introduced in cases that are presumably normal. Everyone knows that the head of the pancreas varies very much indeed in its outline conformation, so that the diagnosis of chronic pancreatitis is not infrequently made. Why? Because we do not find what we expect to find.

Dr. John E. Cannaday, Charleston, West Virginia.—I would like to ask Dr. Watts to tell us what he believes to be the best surgical method of treating typical chronic pancreatitis.

Dr. Watts (closing).—Dr. Hendon asked regarding the state of the gall-bladder in these cases. In all of these 3 cases of what I called subacute pancreatitis I found gall-stones. In fact, I made a diagnosis of gall-stones and found what I went after, but I found something else. I found this condition that Dr. Ransohoff discussed—a variation in the size and consistency of the pancreas. Of course, we were aware of the variations in the size and consistency of the pancreas, but in these cases there was something else besides thickening, and the pancreas presents fat necrosis which undergoes by its presence a more or less acute process. In this connection I want to emphasize the importance of noticing the outlines of the pancreas in these cases as to whether or not these outlines are well defined. In these 3 cases which I reported the outlines of the pancreas were not well defined at all. They sort of shaded off into the peripancreatic tissue. Those are the cases that I wish you men would think about. I have not found any reference to these cases in the literature, although I have not reviewed it exhaustively. I have seen no reference to these cases of subacute pancreatitis which is found in the course of an operation undertaken after a diagnosis of gall-stones. Dr. McGlannan evidently has encountered such cases. How many of these cases have you had, doctor?

Dr. McGlannan.—I can think only of 2 now.

Dr. Watts.—In these cases we find these fat necroses and gall-stones and we drain and are expected to do more and these patients do not do well oftentimes as the result of a flare up.

Dr. Jordan.—Do you think in the acute cases of gall-bladder trouble the subacute process in the pancreas is lighted up?

Dr. Watts.—I call them subacute cases and perhaps the operation itself is a factor in lighting them up, and possibly that would have gone on as the result of self-digestion, the vessel being open and you get a so-called pancreatic apoplexy.

Dr. Cannaday has asked my views with reference to the treatment of chronic pancreatitis. I must say that I cannot speak authoritatively on that subject. Possibly Dr. Le Grand Guerry can tell us more about it. He thinks we should do cholecystenterostomy, while I have done usually cholecystostomy.

TRAUMATIC RUPTURE OF THE SPLEEN

By A. Murat Willis, M.D.
Richmond, Virginia

Subcutaneous traumatic rupture of the spleen is unusual without other serious injuries; especially is this true if the spleen is not markedly enlarged or unduly movable. The 4 cases which I wish to report occurred in our service in a period of approximately three years. The first was on August 24, 1914, and the last October 7, 1917.

Case I.—B. F. V., male, white, aged twenty-eight years, entered the hospital August 24, 1914, with a history of an abdominal injury.

The family and past histories were unimportant.

Personal Inspection. In a runaway accident at night, twenty-four hours before his admission his horse suddenly turned and carried the vehicle up a two-foot embankment. He was thrown against the dash-board (the angle of which pressed sharply under the left costal arch) and out of the buggy. The wheels passed over him, but he was not dragged. Although he suffered with an agonizing pain in the left shoulder and dizziness, he regained his feet and stumbled around looking for his horse for a period of about ten minutes. With the aid of a companion he walked about one mile to his home, where he spent a most uncomfortable night, owing to the agonizing pain in his shoulder, which seemed to be accentuated in a recumbent position. He was thirsty, had profuse sweats and grew excessively weak during the night,

and as the day progressed these symptoms seemed to become worse. He was nauseated about 10 A.M. and vomited (vomitus contained no blood). His urine was very scanty and a catheterized specimen showed a small quantity of blood.

Physical Examination. The patient was seen about twenty-four hours after the accident. At this time his temperature was 96°, pulse 94, regular and of small volume; respirations, 34, short and superficial, and he complained of pain with each respiratory movement. He was pale and had a sharp, pinched expression about the face and nose. The lungs and heart were normal and there were no bruises on shoulders or thorax. The abdomen was slightly rigid and markedly tender, and there was dulness in the left flank. There was no fracture of ribs or other bones.

Hemoglobin, 70 per cent.; white blood count, 26,100.

Urine. Straw color; cloudy; acid; specific gravity, 1020; albumin plus; sugar negative. Microscopic: Numerous pus and red cells were seen; few granular and blood casts.

Diagnosis. Abdominal hemorrhage from ruptured spleen or liver.

Operation. Under cocaine anesthesia twenty-seven hours after injury a left rectus incision was made. On opening the peritoneal cavity a large amount of free blood was encountered. It was thought best to continue the operation under ether anesthesia. The incision was rapidly enlarged and hand passed in the cavity; the spleen was examined and found to be enveloped in large blood clots. This was removed and there was active bleeding. An open rent was felt along the posterior convex surface extending the whole length of the spleen. Owing to the short pedicle we encountered considerable difficulty in making a proper ligation. The patient at this time was in considerable shock. Hypodermoclysis was started and a rapid closure of the wound was then made.

Shortly after operation his condition became so serious that it was thought best to use transfusion. The patient's

mother acted as donor and blood was allowed to flow over for a period of fifteen minutes. Immediate improvement commenced with the introduction of fresh donor's blood into the vein.

The patient reacted well and made a fair convalescence, and was discharged from the hospital five weeks later.

Blood Findings: August 27. Leukocytes, 10,600; hemoglobin, 70 per cent. Differential count: polymorphonuclears, 82 per cent.; lymphocytes, 18 per cent.

September 1. Hemoglobin, 65 per cent.; total reds, 4,800,000.

September 5. White blood count, 35,000; polymorphonuclears, 88 per cent.

September 12. Total leukocytes, 25,000; hemoglobin, 65 per cent.; polymorphonuclears, 86 per cent.

Urine: August 26. Amber color; cloudy; acid; specific gravity, 1022; albumin plus; sugar negative. Microscopic: pus cells and red blood cells.

August 27. Amber color; cloudy; acid; specific gravity, 1018; albumin large amount; sugar negative; abundant pus.

Pathological Report. The spleen, on inspection, was normal in size, color and consistency. On its posterior surface there was a rent extending the full length of the spleen. On the upper border there was a laceration 1 cm. deep and 3 cm. in length.

Microscopic Report. Malpighian bodies and splenic pulp presented a normal picture. There was an increase in connective tissue.

Case II.—Occurred in the service of my associates, Drs. Motley and Smith. C. R., male, white, aged fourteen years, entered the Abingdon Hospital with a history of an abdominal injury.

The family and past histories were unimportant.

Personal Inspection. While coasting he was thrown from his sled and his left lower chest and upper abdomen collided with a large tree, the accident caused considerable pain and

dizziness. He managed to walk across the street to a store and later was taken home in a conveyance. He was seen by a physician almost immediately after the accident, who noted a temperature of 96° and pulse of 90. At this examination he was rigid and tender over the left side of his abdomen. Twenty hours later his temperature was 101° and pulse 140.

Physical Examination. Twenty-seven hours after accident patient was admitted to the hospital with a temperature of 100° and pulse of 140. He complained bitterly of an agonizing pain in the left shoulder. This pain was not accentuated by the movement of the shoulder-joint, nor was there any evidence of trauma. He also complained of pain in his chest and abdomen. Chest examination showed dulness and absence of breath sounds over the lower posterior border of the left lung. The abdomen was flat, except for a slight distention of the epigastrium. On palpation the abdomen was extremely rigid and tender throughout, and on percussion there was dulness in the left flank extending outward as far as the axillary line and upward to the dulness noted in left chest.

The urine was negative. The hemoglobin was 70 per cent. and leukocytes 16,000.

Operation. Under ether anesthesia a high left rectus incision was made. As soon as the peritoneum was opened a large quantity of blood gushed out. The spleen was located, clamps were applied to the pedicle and it was removed. The stumps of the vessels were sutured with No. 4 catgut, the blood was sponged out of the peritoneal cavity and posterior drainage was inserted.

The patient had lost an appalling amount of blood. Soon after operation his condition became so desperate that a direct transfusion was performed by means of Bernheim tubes. A nurse acted as the donor. Almost immediately after the blood began to flow the patient's lips became pink, the color returned to his cheeks and he awoke and began to talk rationally.

The postoperative course was somewhat marred by a pleural effusion on the left side.

Blood Findings: December 18. Leukocytes, 16,000; hemoglobin, 70 per cent.; polymorphonuclears, 88 per cent.; small mononuclears, 7 per cent.; large mononuclears, 5 per cent.

December 21. Leukocytes, 30,000; hemoglobin, 68 per cent.; erythrocytes, 3,500,000; polymorphonuclears, 80 per cent.; small mononuclears, 11 per cent.; large mononuclears, 9 per cent.

December 23. Leukocytes, 21,000; hemoglobin, 65 per cent.; erythrocytes, 4,000,000; polymorphonuclears, 83 per cent.; eosinophiles, 5 per cent.; small mononuclears, 4 per cent.; large mononuclears, 8 per cent.

December 26. Leukocytes, 20,000; polymorphonuclears, 70 per cent.; small mononuclears, 8 per cent.; large mononuclears, 8 per cent.; eosinophiles, 14 per cent.

December 31. Leukocytes, 20,000; erythrocytes, 4,200,000; polymorphonuclears, 75 per cent.; small mononuclears, 10 per cent.; large mononuclears, 1 per cent.; eosinophiles, 14 per cent.

January 5. Leukocytes, 20,000; hemoglobin, 70 per cent.; red cells, 3,800,000; polymorphonuclears, 76 per cent.; small mononuclears, 10 per cent.; large mononuclears, 4 per cent.; eosinophiles, 10 per cent.

January 11. Leukocytes, 16,000; hemoglobin, 70 per cent.; red cells, 3,800,000; polymorphonuclears, 72 per cent.; small mononuclears, 4 per cent.; large mononuclears, 15 per cent.; eosinophiles, 9 per cent.

Pathological Report. The spleen was slightly enlarged for one of his age, measuring 9.5 cm. in length by 7 cm. in breadth by 2.5 cm. in thickness. The color and consistency were normal. On its anterior surface there was a rent extending to the middle of the spleen.

Microscopic Report. The Malpighian bodies were few in number and were pressed apart by an increase of splenic

pulp. This increase was due to an infiltration with leukocytes and lymphocytes.

Case III.—C. P. G., male, white, aged forty-six years, entered the Johnston-Willis Sanatorium November 12, 1916, with a history of an injury in an automobile accident.

The family and past histories were unimportant.

Personal Inspection. He stated that he was injured in an automobile accident which occurred while driving his car over wet roads. He lost control of the car, it turning over and catching him between the steering wheel and seat, injuring him in the abdomen. He was released by his companions and stated that he was seized with violent pains in his left shoulder and under the costal arch of the left side. He was moved to a house near by the roadside, where he stayed for two hours before being seen by a physician.

When first seen in the automobile on arrival at the hospital he was breathing with much difficulty. He had his right hand placed on his left shoulder, stating that he was suffering agonizing pain in this region, and under the left costal arch.

When placed in bed it was found that his pain was greatly increased when he was in the recumbent position.

Physical examination revealed nothing on inspection except that the patient was breathing with much discomfort and a face which showed much anxiety. There was not an abrasion or bruise on the skin surface. His temperature was 96°, pulse 80, respiration 22, skin cold and clammy. He was tender all over the abdomen; very rigid; showed much tenderness in the left costovertebral angle. The pain in the shoulder, of which he complained, was not accentuated by palpation.

Blood findings showed total white cell count 25,200, with a hemoglobin of 90 per cent. at 7 o'clock; one hour later his total white cell count was 23,600 and hemoglobin 70 per cent.

A provisional diagnosis of a ruptured spleen was made from the foregoing data and it was thought best to explore this patient under local anesthesia to confirm the diagnosis.

Operation. On entering the peritoneal cavity through a left rectus incision a large amount of free blood was encountered. We deemed it wise to switch to a general anesthetic. We enlarged the incision, located the pedicle of the spleen, which was shorter than normal, and somewhat adherent by old adhesions. We being unable to get free access to this organ our incision was enlarged by making a left transverse incision. The pedicle was ligated, the ruptured spleen removed and all the free blood removed with the suction apparatus. Closed the wound with drainage. Hypodermoclysis was given on the table. Stomach was washed and a large amount of dark green fluid was obtained.

A transfusion was advised by my associate, Dr. Budd, but he was unable to match his blood with the donors, among several of which was his son. Patient reacted well and seemed to improve for the next few days.

Blood findings the next day, November 13, was as follows:

Total white cell count, 17,600; hemoglobin, 70 per cent.; polynuclears, 70 per cent.; small lymphocytes, 16 per cent.; large lymphocytes, 14 per cent.

November 14. Leukocytes, 10,800; hemoglobin, 70 per cent. Differential not made.

November 15. Leukocytes 8000; polynuclears, 66 per cent.; small lymphocytes, 12 per cent.; large lymphocytes, 22 per cent.; hemoglobin, 68 per cent.

November 16. Leukocytes, 7200; hemoglobin, 60 per cent.

November 19. Leukocytes 7000; polynuclears, 88 per cent.; lymphocytes, 12 per cent.; hemoglobin, 60 per cent.

Pathological Report. Spleen was not enlarged; color and consistency normal. There was a stellate rent on the anterior surface of the spleen covering a little over half of the surface.

The patient was given continuous saline, nothing by mouth, hypodermoclysis and morphin, gr. $\frac{1}{6}$, every four hours when necessary.

His stomach was washed repeatedly, which always brought back a good deal of dark green fluid. He suffered a great deal from distention, which we were unable to relieve by the ordinary methods.

Patient's condition seemed about the same for the next seven days, but on the eighth day he began to suffer more than usual with distention; the pulse became weak and rapid and on the eighth day he died. No autopsy.

Case IV.—F. C., male, colored, aged fifteen years, entered the hospital September 12, 1917, with a history of an injury in an automobile accident.

Personal Inspection. First seen by his family physician about one hour after the accident. At the time he was unconscious and showed symptoms of concussion of the brain and those of an abdominal injury.

Physical Examination. Three hours after the accident the patient was admitted to the hospital in a semiconscious condition. There was a slight laceration of the scalp over the right parietal region. There was no other evidences of injury on other portions of the body. His temperature was 98.4°, pulse 74, respiration 26. Reflexes normal; eye-grounds normal; consciousness returning. Chest negative. Upon examination the abdomen was slightly distended; slight rigidity and tenderness over the entire abdomen, increased over left rectus muscle.

The urine was negative.

Blood on admission: Leukocytes 22,800; polynuclears, 81 per cent.; lymphocytes, 19 per cent.; hemoglobin, 90 per cent. Blood examination two hours later: Leukocytes, 22,000; polynuclears, 86 per cent.; lymphocytes, 9 per cent.; large mononuclears, 5 per cent.; hemoglobin, 70 per cent.

Abdominal pain increased, pulse rose to 116, temperature 100.4°, respiration 40; hemoglobin 70 per cent.

Diagnosis. Internal hemorrhage from rupture of liver or spleen.

Operation. Twenty-four hours after the accident the abdomen was opened through a median incision, extending from the costal arch down four inches. The peritoneal cavity contained a large amount of blood. The liver was thoroughly explored for fracture, but no injury could be discovered. The spleen was next palpated and evidences of injury could be made out, although the incision did not permit a satisfactory examination. A transverse incision was then made through the abdominal wall from the midpoint of the first incision to just beneath the left costal arch. There was a blood-clot adherent to the spleen, which on further examination showed two lines of fracture. The injury was confined to the inferior half, the upper line being just beneath the center of its longitudinal axis. The organ seemed to be somewhat enlarged and had only a slight range of movement on account of either very short ligaments or else anomalous fixation bands. It seemed to be firmly fixed and some little time was required to mobilize it sufficiently to permit a ligation of the vessels. This was done with No. 2 tanned catgut. The wound was closed in layers in the usual way, without drainage. The patient stood the operation fairly well and was given subcutaneous saline, leaving the table with a pulse of 130. Postoperative convalescence was satisfactory.

Blood Findings: September 19. Leukocytes, 8400; hemoglobin, 65 per cent.; red cells, 4,320,000. In the course of the next ten days hemoglobin fell to 60 per cent., and then slowly increased, reaching 70 per cent. three weeks after operation; white cell count at that time was normal.

In summing up the histories of these 4 cases we find that all present the following points in common:

1. They were all males.
2. Blow on left side of body in splenic region.
3. Secondary anemia and leukocytosis.
4. No external evidence of injury about the body.
5. Rigidity and tenderness of the abdomen.

6. All had shock following splenectomy.

7. All the spleens were practically normal in size with short pedicles and without any history of previous disease of the organ.

Three out of the four had agonizing pain in the left shoulder which was promptly relieved by splenectomy. The fourth case was a child sent in for concussion of the brain, and as he was in a semiconscious condition we were unable to elicit a satisfactory history.

Three were operated on twenty-four hours after the injury.

In 2 of the cases a marked increased leukocytosis persisted after the operation over a period of from two to four weeks.

Three cases had satisfactory convalescence. One died eight days after operation.

The fatal case showed a continuous fall in hemoglobin and a decreasing leukocyte count.

In the first 2 cases operation was followed by direct transfusion, with apparently good results.

Some years ago in a patient on whom we had done a splenectomy for Banti's disease there was a postoperative bleeding from the pedicle and a second operation was necessary to control hemorrhage. The patient reacted very well from this operation, but died a few days later with symptoms suggesting severe anemia. At autopsy no internal hemorrhage was found and we did not believe that a sufficient amount of blood had been lost from the accident to account for the fatality. Since the amount of hemorrhage found in the cavity at the time of the second operation was not sufficient to have caused death, we felt that in some way the removal of the spleen plus hemorrhage had produced a severe secondary anemia that would not have resulted from the loss of the same quantity of blood from any other abdominal organ. We were strengthened in this belief by the experimental findings of Musser,[1] who says: "In the dog splenectomy is followed by a decrease in the red blood cells and hemo-

globin reaching the lowest level about the twenty-sixth day, the hemoglobin varying from 50 to 65 per cent. and the red cells from 2,800,000 to 3,500,000. From this time to the eighty-second day a constant increase in hemoglobin and red cells occurs, the animal not reaching or approximating its normal level, however, until the end of about four and a half months." Pearce and Austin,[2] also experimenting with dogs, showed "that in the absence of the spleen the function of forming red blood corpuscle-phagocyting cells, normally a minor activity of the lymph nodes becomes highly developed in the latter organs, and that these cells and the stellate cells of the liver thus assume, in part at least, the function of destroying red blood corpuscles by phagocytosis."

Having in mind the former unfortunate experience in the case of Banti's disease and also the results of Musser, Pearce and Austin just quoted, we promptly resorted to transfusion in our first 2 cases of splenic rupture with most gratifying results. Since, however, our experience with splenectomy following hemorrhage was small, we determined to study the points which had come to light in these cases with the aid of animal experimentation.

The points we wished to investigate were these: (1) What effect has simple splenectomy upon the blood picture? (2) What change is produced when splenectomy is performed following a considerable hemorrhage, as was the case in our patients with ruptured spleen? (3) In what way does transfusion modify the results of splenectomy? Our experiments may be grouped under three heads: (1) Splenectomy with no loss of blood. (2) Splenectomy with loss of a considerable amount of blood. (3) Splenectomy with loss of blood and subsequent transfusion.

In our experiments on dogs conducted by Dr. S. W. Budd and myself in 1914 and 1915 the abdomen was opened, under ether anesthesia, by a high left rectus incision, and the spleen delivered through the wound. Ligatures were placed around the splenic artery and vein and also around the entire splenic

ligament. In groups II and III the ligatures around the splenic artery and vein were not tightened until the animal had lost blood enough to reduce the hemoglobin to 70 per cent. Indirect transfusion was used in group III, blood being introduced through the splenic vein. In order to simulate as nearly as possible a ruptured spleen we macerated the organ and squeezed part of the splenic pulp into the abdominal cavity in all experiments.

Under group I, splenectomy with no loss of blood, eighteen dogs were used. The majority of these showed but a slight decrease in hemoglobin; a small number showed no loss; only a few showed a marked decrease to 60 per cent. in the first few weeks with a gradual return to normal.

In group II, splenectomy with dogs bled until the hemoglobin registered approximately 70 per cent., fifteen dogs were used. Most of these showed the same blood picture as the last cases in group I, which was a gradual decrease to 60 per cent., then a slow rise. One case showed a marked anemia and died on seventh day. Two others developed a grave anemia, hemoglobin going as low as 45 per cent. followed by a gradual rise.

In group III, splenectomy animals bled until hemoglobin reached 70 per cent. followed by transfusion. The blood picture in this series of cases was similar to the majority of group II. In this series five dogs were used.

The blood findings in our human cases that recovered simulated the majority of those in groups II and III, that is a gradual decrease in hemoglobin as low as 55 per cent., then a return to normal. The experiments just detailed seem to establish the fact that the destruction of red cells following a simple splenectomy is by no means excessive. A few of our animals, however, showed a marked secondary anemia following splenectomy after hemorrhage.

It will be observed that the studies of the blood in these cases were not elaborate. From previous experimental work it seemed safe to assume that the anemia present was

of the secondary type and that hemoglobin estimation represented a fair index of its degree.

We wish to call attention especially to pain in the left shoulder as a symptom of considerable diagnostic importance. This pain is undoubtedly referred from the diaphragm and is due to subphrenic irritation from blood clots around the spleen. Pain of a similar nature in cases of diaphragmatic pleurisy has been referred to by Capps,[3] who states "that the central portion of the diaphragm receives it sensory nerves from the phrenic nerve, which carries efferent impulses to the cervical cord, and sets up reflex pain in the corresponding spinal segments, namely, the third and fourth cervical."

From the foregoing observations we are led to the belief that in certain cases of splenectomy following hemorrhage, death occurs as the result of a severe secondary anemia out of proportion to the amount of blood lost and probably incident to the abscess of the spleen.

Fauntleroy, A. M.[4] *Traumatic rupture of spleen (complete); splenectomy.* He complained of a curious, deep-seated pain in left shoulder, which was not increased by movement of the joint, and, as examination showed no contusion or dislocation, it was thought to be a slight sprain in this region, due to an effort to catch himself in falling. It is to be noticed, however, that this pain disappeared completely after operation, and it is possible that it has some diagnostic value when the spleen is traumatically involved.

References

1. Musser, J. H., Jr.: An Experimental Study of the Changes in the Blood following Splenectomy, Arch. Int. Med., 1912, ix, 592.

2. Pearce, R. M., and Austin, J. H.: Changes in the Endothelial Cells of the Lymph Nodes and Liver in Splenectomized Animals Receiving Hemolytic Serum.

3. Capps, Joseph A.: Am. Jour. Med. Sc., 1916, cli, 333.

4. Fauntleroy, A. M.: Ann. Surg., 1913, lvii, 68–71.

DISCUSSION

Dr. Edward H. Ochsner, Chicago.—The author of this paper brings up a number of very interesting questions, one of which is: Why do some of these patients and dogs stand splenectomy so well, and why do others in the course of a number of weeks develop severe anemia and then die? The question which arises in my mind is this: Is this difference in the reaction to the operation possibly due to the fact that some of these patients and some of these dogs have supernumerary spleens and others not?

I believe the large majority of individuals have supernumerary spleens and that these will stand splenectomy, while others who have no supernumerary spleens will succumb. I believe a little spleen tissue is absolutely essential to life. Further observations along this line would be of great value.

Dr. F. G. Du Bose, Selma, Alabama.—I would like to ask the essayist whether there was enlargement of the lymph glands in his series? I saw one patient with a rupture of the spleen who, after the removal of his spleen, developed a general lymphadenitis; otherwise his blood and general condition were really improved by the operation. The blood was carefully examined every two weeks for a period of three or four months, but he did develop lymphadenitis which subsided within a year.

Dr. Willis (closing).—Two of our cases were observed long enough for the blood to return to normal. There were no lymphatic enlargements. I feel sure that all of these cases do not react alike, and that Dr. Ochsner's suggestion of an accessory spleen is along the right line. In looking up the literature on traumatic rupture of the normal spleen, we have found about 250 cases with a mortality of between 40 and 50 per cent. Some died soon after the operation, and some lived several weeks.

From our experimental work on dogs and from our observation on human cases we are satisfied that the death can be explained in no other way except along the lines Dr. Ochsner suggests.

RECONSTRUCTION OF THE CHOLEDOCHUS

By Le Grand Guerry, M.D.
Columbia, S. C.

Surgeons are occasionally forced to the necessity of reconstructing the common duct. Surgical literature is full of case reports, and many methods have been advocated by equally as many writers—transplanting of fascia; transplanting of bloodvessels; transplanting of the peritoneal layer of the duodenum, and so on to the end of the chapter. Some of the operations are good and some are distinctly bad.

Experimentally many plans have been tried on dogs with varying degrees of success. Unfortunately what will work in the dog laboratory will not always work in the human abdomen, and the simple method that we present, although done independently under the force of circumstances seven years ago at our clinic, is not an original plan, for I find in reviewing the literature case reports of almost the identical procedure.

In January, 1916, Dr. W. J. Mayo published a splendid report on what is virtually the same operation. It appears to me that in our effort to find something new under the sun we have focussed our thought too much on the experimental laboratory and have overlooked the most natural way of overcoming these defects in the bile passages.

Some of the reasons why it is necessary to reconstruct the common duct are as follows:

Reason I. In cases of permanent obstruction at the

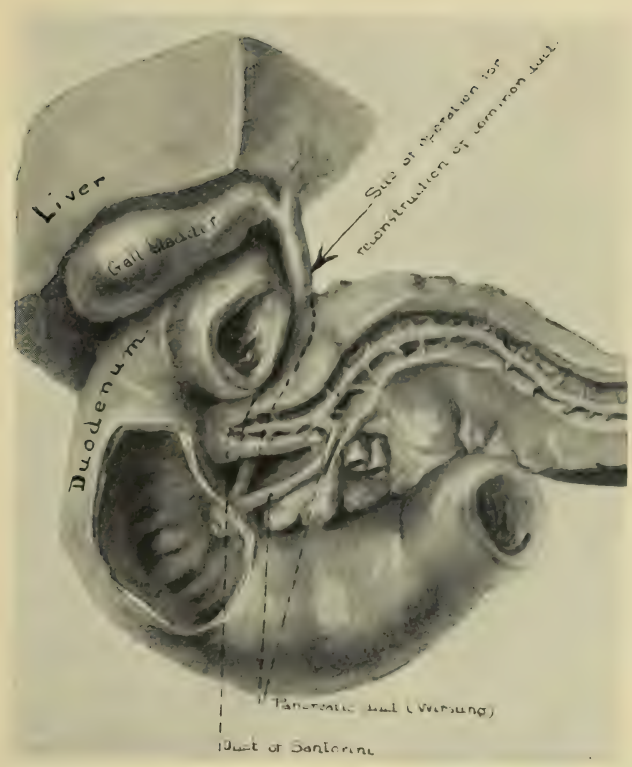

Fig. 1.—Drawing modified from Spalteholz.

Fig. 2

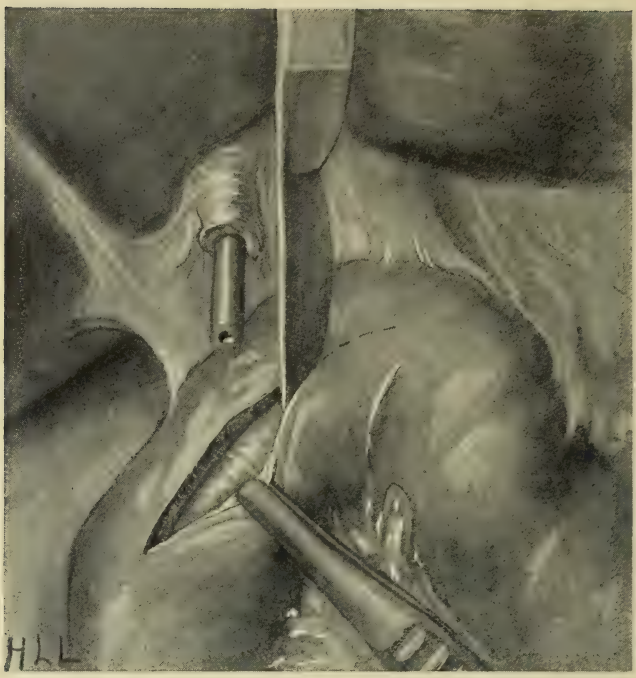

Fig. 3

Fig. 4.

Fig 5

Fig. 6.

Fig. 7

head of the pancreas, if the gall-bladder is intact we will have plain sailing. Cholecysto-duodenostomy is, however, a short-circuiting operation rather than a reconstructing of the bile passages; if the gall-bladder has been removed there will be another story to tell.

Reason II. It may be necessary to restore the bile passages on account of inflammatory stricture of the common duct. If the stricture of the common duct extends above the junction of the cystic and hepatic ducts it may be necessary to excise the strictures and then apply if possible the method to be described.

Reason III. The common duct may be divided in the operation of cholecystectomy. As a matter of fact, injuries to the choledochus are usually the result of operative measures. If the accident is discovered immediately, repair is much easier than if a secondary operation is necessary to correct the injury. It is vitally necessary to remember here that the junction of the hepatic and cystic ducts which form the common duct is not always at a fixed point.

Whenever we attempt the operation of cholecystectomy the point just mentioned must be constantly borne in mind. The usual position at which the cystic duct enters the hepatic duct is about three-quarters of an inch below that portion of the hepatic duct which lies within the liver itself. However, the junction of these two ducts which form the common duct may occur as high up as the fissure of the liver, or they may run parallel with each other and unite almost at the entrance into the duodenum; according to Gray's *Anatomy*, they may remain independent throughout their course, or the junction may occur at any point between the fissure of the liver and the duodenum.

Not only then do we have the junction of the hepatic duct and the cystic duct at a varying point, but the two ducts running a parallel course and in intimate association one to the other and at times overlapping each other. When this unusual anatomical situation occurs, unless the two ducts

are isolated and identified before the gall-bladder is removed, accidental injury to the hepatic or common duct can and often does occur.

A case within the last few weeks beautifully illustrates this point: A male patient, almost sixty years of age, with a large stone in the ampulla of Vater, producing obstruction of the common duct. The hepatic and cystic ducts were both distended to the size of one's index finger and could be clearly traced almost to the duodenum before the junction was effected.

The object of this short paper, however, is not to discuss the general subject of injuries to the choledochus but to report briefly a method of reconstruction that we have used in 7 cases. What we seek to do is to form a direct union between the hepatic duct or the common duct and the duodenum. The fundamental thing in the plan, as the illustrations will show, is to so mobilize the mucosa of the duodenum that when the suture line is completed the mucosa of the duodenum will be directly united to the mucosa of the hepatic duct. If this is done we will have a continuous epithelium-lined passage, and contraction in all probability will not occur.

This point illustrates the inherent weakness in many of the so-called autoplastic reconstructions of the bile passages, and the various operations break down just here. They fail to provide a continuous mucous-lined passage.

Let us thoroughly understand my contention. We are not claiming that this is the only way or even the best way, but we do submit that the plan has a sound surgical and physiological basis.

DISCUSSION

Dr. J. Shelton Horsley, Richmond, Virginia.—In the last three or four years I have done some experimental work in reconstructing the common duct. I tried a method like the

Roux gastro-enterostomy pictured by Moynihan, who recommends it. I would not doubt anything he says, but facts are facts, and it is possible that this operation is successful in the human being, but it does not work in dogs. First of all, there is no pathology in the gall-bladder to work on. The gall-bladder is very thin in dogs, and we find in the dog that the bowel is much thicker than it is in the human being, and in spite of careful suturing it pulls loose from the gall-bladder, and there is leakage. Practically all my dogs died as the result of leakage. We abandoned that method, and I would not care to undertake it on a patient. I then thought of transplanting a vein, as I was working on bloodvessel-suturing at that time. Anyone who has attempted to suture bloodvessels knows that it is a difficult matter to prevent the endothelium from becoming adherent. The object of suturing the bloodvessels is to keep the lumen open, and the slightest rough work will make the endothelium adhere. It is to be expected, with bile going over it, or long before the bile gets a chance to flow over it, the vein will become a fibrous cord. We turned it wrong side out, made the adventitia inside and endothelial layer outside. This is better tissue fascia because it is a tube, and the endothelium being on the outside will adhere to the peritoneal endothelium, and you can obtain more nourishment for the delicate segment of vein. I tried that. I thought it would be superior. It worked for three or four weeks. Later on I saw Dr. Dean Lewis, of Chicago, who does excellent work by using fascia for the construction of a tube. He was enthusiastic about this work, but all of his dogs were killed inside of a month. The longer my dogs lived the worse they did. After I got the dogs so that they would live for a few weeks I killed some of them and secured specimens of the transplanted veins and found that the mucosa had grown over the transplanted segment of vein in places, but if the dogs were permitted to live for a month or more they became sick and shortly died. The last one lived three months and died. The animal lost weight and appetite, vomited occasionally, became very much emaciated and jaundiced, and postmortem examination showed the vein was occluded. After I had gone over the whole thing, I found it was not mucosa so much as it was the submucosa. If you have a burn you cannot apply a Thiersch graft successfully to prevent contraction. If you want to prevent contraction, you must transplant whole skin and get corium as well as the epidermis. A transplanted

vein is foreign tissue, and tends to contract, and sooner or later, if the dog lives long enough, becomes occluded even though epithelium grows over its internal surface. Invariably contraction will take place because of the fact that we have no true corium or submucosa under the mucosa.

To come down to the practical point of what Dr. Guerry said, instead of having an epithelial lining, we must have what corresponds to the corium as well, and Dr. Guerry has illustrated that point. We must have mucosa with what corresponds to the corium and carry that up to the mucosa stump of the duct.

LONG RESECTIONS OF THE INTESTINE

By J. E. Cannaday, M.D.
Charleston, West Virginia

Long resections of the intestine are occasionally necessitated by such conditions as extensive gunshot wounds, gangrene and ileocecal tuberculosis, with multiple strictures. The small bowel varies in length from 15 feet 6 inches to 31 feet 10 inches, the average length being greater in the female. The large intestine varies in length from 3 feet 3 inches to 6 feet 6 inches. People such as the Russian peasantry, who have for generations largely subsisted on a coarse vegetable diet, have longer intestines than those who live on a more concentrated dietary. The removal of the large bowel seems to interfere but little with the bodily nutrition, but after the removal of any considerable length of the small bowel the patient's physiological resources must be very carefully husbanded. Kukula[1] made careful and exhaustive experimental long resections on animals to show by deduction about what proportion of the human intestine could be safely removed.

Both Park[2] and Moynihan[3] have at different times collected all reported cases of long resections of the small bowel, and the total number is not large. It has been rather generally agreed that at least half of the length of the small intestine may be removed, and the patient survive and exist in a fair state of health thereafter. Toombs[4] mentions a case of long resection in which the cecum and adjacent small bowels were

removed. This patient afterward passed through a typical attack of typhoid fever, indicating that Peyer's patches are not necessarily a focus of the disease. After cutting down the length of the small bowel the wastage of fats and albuminous materials in the nutrient must be very great, and this part of the diet must be increased for these cases.

Case Report.—Referred by Dr. D. G. Preston, Burnwell, W. Va. Adult, male, aged thirty years. Weight, 128 pounds.

General History. Abdominal pain over a period of eight months. Gradual loss of weight and strength; moderate constipation; no attacks of diarrhea. Pain beginning in the region of the appendix. Occasional vomiting and considerable regurgitation of food for the past two months. Abdomen slightly rigid, visible peristalsis, some abdominal tenderness. Wassermann negative. White blood count, 6800; pulse, 98; temperature, 99°. Small area of dulness in right upper apex. Diagnosis: Ileocecal tuberculosis causing partial obstruction of small bowel.

Operation. Assisted by Drs. B. S. Preston and Atlee Mairs the following operation was performed January 4, 1916: Right rectus incision about 20 cm. in length. The cecum was an enlarged mass about the size of a grapefruit. Numerous (eight) tubercular strictures of the small bowel were noted. To eliminate these it was necessary to resort to the radical operation as advocated by Caird[5] and resect 300 cm. (10 feet) of the small bowel as well as the cecum and 20 cm. (8 inches) of the ascending colon. A lateral anastomosis was made with the transverse colon. Patient reacted fairly well, but vomiting was quite persistent. Thirty-six hours after the operation it was deemed advisable to reopen the abdomen for the relief of obstruction. Some milky fluid was noted in the right flank. Some of the peritoneal sutures of fine silk had failed to hold. All such points were reinforced with additional silk sutures. A slightly adherent kink was noted in the small bowel, near its junction with the colon. This was straightened out and the contents of the rather

distended small bowel were milked into the colon. Two medium-sized rubber cigarette drains were inserted in the wound. In exactly twenty-four hours the patient vomited again, this time a distinctly dark brown vomit. Under novocain-suprarenin anesthesia an incision was made in the left rectus, a distended loop of small bowel was drawn out and a medium-sized soft-rubber catheter was purse-stringed into this, after the method of Dr. J. W. Long, and brought out through the upper angle of the wound. Bowel contents drained from the tube quite freely. The patient did not vomit again. The tube came away the eighth day. The discharge from the fistula caused some skin excoriation, but the sinus closed in two weeks. After this the patient made an uneventful recovery, and at the end of the fourth week left the hospital by his own request, with his wounds entirely healed. A few days after the operation his bowels began to move, and at first he had an average of six bowel movements every twenty-four hours. This condition gradually improved, and by the time he left the hospital he was having an average number of three movements in twenty-four hours. After his recovery and discharge from the hospital this patient was exhibited before a regular meeting of the Kanawha Medical Society.

X-ray examination by Dr. W. R. Hughey shows a rapid filling of the very limited small bowel, with early entry into the transverse colon, part of the contents regurgitating into the remains of the ascending colon and hepatic flexure. Several negatives were made, the proofs of which are exhibited.

Later History. This patient left the city in severe winter weather and visited friends in the mining regions nearby. During this time he celebrated by getting drunk. A few days later he returned to Charleston, suffering from a very severe cold. He rapidly became worse and developed the symptoms of an acute tuberculosis of the lungs. He reëntered the hospital February 23, 1916, and on February 28, 1916, died.

Postmortem examination made by Drs. M. I. Mendeloff, H. D. Hatfield and the writer. The abdomen was opened by a long, median incision. With the exception of a few adhesions in the location of the former drainage tubes, the intestines were in excellent condition. The anastomosis was as smooth as if it had existed for years. There were no visible signs of visceral tuberculosis. The few palpable mesenteric glands that were present at the time of the original operation had disappeared. The small intestine up to the duodenojejunal junction was measured and found to be only 5 feet 7 inches in length. Of the large bowel there were 4 feet 9 inches.

Fantino[6] suggests that in cases of extensive resection a vicious circle should be established in the remaining segment of bowel in order that the intestinal contents may circulate long enough to complete the digestive and assimilative processes.

Turck[7] says: "A review of the literature and a study of case histories point plainly to the fact that, from a viewpoint of ultimate results, short-circuiting, whenever possible, is much to be preferred to resection, particularly if the lower ileum is involved. In the case reported above, while circuiting was decided against at the time of operation because of the indescribable adhesions, the foul sinuses and fistulous tracts, it is probable that a better ultimate result would have been attained had the worst segments been removed, and the remaining adherent gut shortcircuited in one or two places. This patient certainly has not enough small intestine to permit of thorough absorption, though compensatory intestinal hyperplasia provided adequate digestion."

Henson[8] in his report on Turck's case says that with the usual hospital diet the patient has about six stools per day. He was then given astringent and digestive ferments, but these had no effect whatever on the number of stools.

Henson's summary is so complete that it is quoted along with his tables.

The following table shows the average number of stools per diem under a variety of restricted diets:

Special diet.	Average number of stools per diem.
Chicken, roast beef, toast and cereals	5.00
Oatmeal, sugar and milk	4.25
Irish potatoes, oatmeal, sugar and milk	4.00
Regular hospital diet excluding all meats	3.66
Hospital soft diet	3.00

"The feces were examined after having been on the regular hospital diet for several days, resulting in the following findings: Stool, fluid; quantity, 700 grams; color, light yellow; odor, not especially offensive. Reaction, acid to both litmus and phenolphthalein. Chemical tests for starch, erythrodextrin, sugar, and occult blood were all negative; for mucin, only slightly positive. The stool macroscopically showed a moderate lientery, which, upon microscopic examination, was found to be due to an azotorrhea. The fibers were mostly digested to the point that the transverse striations and nuclei were not shown, but in some both longitudinal and transverse striations could be observed. Steatorrhea not present, nor were crystals of any form in excess.

"The patient was then placed upon the following diet:

	Grams.	Albumin, per cent.	Fat, per cent.	Carbo-hydrates, per cent.	Calories.
Zwieback	100.00	8.00	1.00	75.00	349.60
Toast	200.00	14.00	1.00	110.00	517.70
Potatoes	192.00	2.60	.....	38.40	168 10
Eggs (4)	.....	28.20	22.00	.....	320.92
Butter	30.00	.....	23.00	.....	214.66
Cream	128.00	4.62	34.24	4.50	356.96
Milk	256.00	8.96	6.40	9.98	137.37
Sugar	40.00	.14	.....	37.33	164.00
	946.00	66.52	87.64	275.21	2,229.31

Weight of patient, 63 kilos; 35 grams to kilo equals 2205 equals required calories at rest.

"It is worthy of note that during all preceding periods in which his urine was examined for indican this constituent

was constantly and markedly in excess, but that two days after being on the foregoing diet the indicanuria almost entirely disappeared. While on the diet tabulated his average urinary output was 456 c.c. per diem, the specific gravity of a twenty-four-hour specimen was 10.28, the total urea output in the twenty-four hours was 7.84 grams. His stools following the use of this diet averaged three per diem. The feces within a short time became semiformed with an average weight of 755.2 grams. There were no chemical or microscopic findings different from what has already been recorded while the patient was on the regular diet, with the exception, of course, that the azotorrhea was not present. Further tests for starch, erythrodextrin and sugar were all negative. In this case it would seem probable that a compensatory hyperplasia of the small intestine and a compensatory production of the ferment enterokinase was at least commenced. We know that amylopsin and steapsin are capable of exercising their functions with little auxiliary aid from this ferment. It is reasonable, therefore, to presume that the compensatory production of enterokinase in this patient has reached the point where, together with the enzymes of the pancreatic juice, it is sufficient to digest pretty thoroughly the intake of carbohydrates and fats, but that this compensatory production has not reached the point of thoroughly digesting proteids.

"For the purpose of demonstrating to what extent the absorbability of the intestine is interfered with, Sahli's method was tried, the patient being given 0.15 gram iodoform in a salol-coated gelatin capsule with an Ewald test meal. The appearance of the iodin reaction in the saliva on two different occasions was delayed six hours, hourly tests being conducted."

I append a table of cases taken in part from Park, Moynihan and other sources, and relatively brought up to date.

Operator and reference.	Length of section of intestine resected.	Result.
Schlatter: Kor.-Bl. f. Schweiz. Aerzte, 1899; Beiträge z. klin. Chir., 1906, xlix, i; Lancet, 1900, i, 207.	192 cm. after "considerable shrinking" subsequent to removal.	Recovery.
Boeckel I.: Bull. de l'Acad. de méd., Paris, 1912, lxviii, 241.	200 cm.	Recovery.
Lexer: Berl. klin. Wchnschr., 1900, xxxvii, 4; Albu: Berl. klin. Wchnschr., 1901, No. 50.	200 cm. small intestine.	Recovery.
Peterson: Ref. Rusch-haupt., Inaug. Diss., Bonn, 1901.	202 cm.	Recovery.
Flint, J. M.: Johns Hopkins Hosp. Bull., 1912, xxiii, 127.	204 cm. collapsed.	Death six days later.
Reder, Francis: Personal communication.	204 cm. ileum.	Recovery.
Koeberle: Centralbl. f. Chir., 1881, viii, 249.	205 cm. small intestine.	Recovery.
Myaki: Arch. f. klin. Chir., 1910, xciii, 768.	206 cm. resected.	Death four days later of septic peritonitis.
Enderlin: Ref. Lauenstein: Deutsche Ztschr. f. Chir., 1909, c, 169.	207 cm. resected.	Recovery.
Kocher, quoted by Trzebicky: Arch. f. klin. Chir., 1894, xlviii.	208 cm. small intestine.	Recovery.
Mikulicz: Rothe, Beiträge z. klin. Chir., 1902, xxxiii, 140.	215 cm.	Recovery.
Dreesmann: Berl. klin. Wchnschr., 1899, xxxvi, 337.	215 cm. ileum.	Recovery.
Axhausen: Mitth. a. d. Grenzgeb. d. Med. u. Chir., 1909, xxi, 55.	215 cm.	Recovery.
Karlow: Hygeia, No. 3.	215 cm. small intestine.	Recovery.
Thon: Deutsche med. Wchnschr., 1909, xxxv, 742.	220 cm.	Recovery.
Dekonski: Ref. Istomin: Russ. med. Rundschau, 1910, viii, 329.	220 cm.	Recovery.
Kouwer: Nederl. Tijdschr. v. Geneesk., Amst., 1898, 2 R., xxxiv, d. 2, 887.	224 cm.	Recovery.
Shepherd: Centralbl. f. Chir., 1898, xxv, 397.	234 cm. ileum.	Recovery.
Kukula: Arch. f. klin. Chir., 1900, lx, 887.	237 cm. ileum.	Recovery.
Harris: New York Med. Rec., 1902, lxii, 566.	239 cm. ileum.	Recovery.
Kirkwood, W. L.: Medical Journal of Australia, 1915, ii, 49.	240 cm. resected.	Recovery; abscess; second operation for recurrence of symptoms 9 months later. Recovered; well and better than after first.
Hayes, quoted by Harris.	248 cm. small intestine.	Recovery.
Brenner and Denk: Wien. klin. Wchnschr., 1907, xx, 1649.	250 cm.	Recovery.
Enochin: Red. Istomin, Russ. med. Rundschau, 1910, viii, 329.	250 cm.	Recovery.
Peck, quoted by Harris.	251 cm. small intestine.	Recovery.
Blayney: British Med. Jour., 1901, ii, 1456.	255 cm.	Recovery.
Lauwers: Ann. de la Soc. de Chir. (de Belge), 1901, ix.	265 cm. small intestine.	Recovery.
Park: Arch. Onter. de Chir., i; Centralbl. f. Chir., 1904, xxxi, 55; Buffalo Med. Jour., 1903.	265 cm. small intestine.	Recovery.
Payr: Arch. f. klin. Chir., 1902, lxvii, 181.	265 cm. ileum.	Recovery.
Pokotilo: Chirurgia, 1909, xxv, 169.	270 cm. resected.	Recovery.
Mitchell, quoted in Kelly and Noble's Abdominal Surgery, Philadelphia, 1908, p. 442.	274 cm.	Death.
Kopfstein: Revue de Bohm. Med., 1909, Ref. Wien. Med. Bl., 1910.	275 cm.	Recovery.
Maydl, quoted by Kukula: Arch. f. klin. Chir.	284 cm. ileum and 8 cm. colon.	Recovery.

Operator and reference.	Length of section of intestine resected.	Result.
Childe: British Med. Jour., 1901, ii, 891; Practitioner, 1909, cxxxii, 364.	289 cm.	Recovered; lived seven months.
Lorenz: Wien. klin. Wchnschr., 1906, xix, 610.	292 cm.	Recovery.
Goebell: Deutsch. Ztschr. f. Chir., 1905, lxx, 608.	300 cm. small intestine.	Recovery.
Fantino: Riforma med., Naples, 1902, xviii, 181.	300 cm.	Death one month after operation.
Tombs, quoted by Murphy: Year Book General Surgery, 1915.	305 cm.	Recovery.
E. Staehlin: Annals of Surgery, 1907, xlv, 49, and personal communication.	305 cm.	Recovery.
Fantino: Gaz. medica di Torino, 1896, xlviii.	310 cm. ileum.	Recovery.
Monprofit: Rev. di Chir., 1899, xx, 579.	310 cm. intestine (230 cm. ileum 80 cm. large intestine).	Recovery.
Ghedini: Clin. Chir., Milano, 1905, xii, 278.	315 cm.	Recovered; lived three and one-half months.
Zusch: Deutsch. med. Wchnschr., 1909, p. 739 (operation in 1896).	316 cm.	Recovery.
Fischer, A.: Orvosi hetil, No. 37, 1905; Centralbl. f. d. Grenzgeb. d. Med. u. Chir., 1906, ix, 70.	316 cm.	Recovery.
Zeidler: Centralbl. f. Chir., 1906.	318 cm. ileum.	Recovery.
Whitall: Annals of Surgery, 1911, civ, 699; Annals of Surg., 1913, lviii, No. 5.	320 cm.	Recovery.
Norton: Ref. Kelly and Noble, Abdominal Surgery, Philadelphia, 1908, p. 442.	322 cm.	Recovery.
Myaki, quoted by Flint: Johns Hopkins Hosp. Bull., May, 1912.	328 cm. ileum, 225; colon, 13.	Recovery.
Ruggi: Policlinico, 1896; Centralbl. f. Chir., 1896, xxiii, 365.	330 cm. principally ileum.	Recovery.
McGuire: Surg., Gynec. and Obst., January, 1913.	336 cm.	Recovery.
F. J. Brougham: Surg., Gynec. and Obst., 1907, v, 782.	345 cm.	Recovery.
Nicola: Clin. Chir., 1910.	350 cm.	Recovery.
Baracz: Arch. f. klin. Chir., lxxxiii, 478.	350 cm.	Death two days after operation.
Von Eiselsberg (the case is given by Park, Payr and others, but I can get no further particulars).	350 cm.	Death after twenty five days.
J. W. Long: Personal communication.	360 cm. ileum.	Death.
Obalinski: Arch. f. klin. Chir., 1894, xlviii, 16.	365 cm. ileum and cecum.	Death in twenty-eight hours.
A. Werelius: Jour. Am. Med. Assn., 1907, xlviii, 945.	12 feet two inches (365 cm.).	Recovery; button passed on tenth day.
Mitchell: British Med. Jour., 1902, ii, 975.	366 cm.	Recovery.
Turck: New York Med. Jour., 1914, xcix, p. 316.	371 cm. ileum and colon.	Recovery.
W. D. Haggard: Personal communication.	380 cm. ileum and cecum.	Death in eleven days.
Pauchet: Personal communication; Jahresbericht f. Chir., 1905, xi, 732.	400 cm. ileum.	Recovery.
Friedrich: Med. Klin., 1904–05, i, 25.	400 cm.	Recovery.
Stolz: Deutsch. med. Wchnschr., 1909, p. 744.	475 cm.	Recovery.
Axhausen: Mitt. a. d. Grenzgeb. d. Med. u. Chir., 1909, xxi, 55.	475 cm.	Recovery.
Storp: Deutsch. Ztschr. f. Chir., 1907, lxxxvii, 322.	510 cm. small intestine (the entire ileum and part of the jejunum).	Recovery.
Nigrisoli: Atti del. xvi Congr. Ital. de Chir., 1902.	520 cm. small intestine.	Recovery.
Ghedini: Clin. Chir., Milano, 1905, xiii, 278.	524 cm.	Recovery.
Brenner, reported by Denk: Mitth. a. d. Grenzgeb. d. Med. u. Chir., 1910, Heft i, p. 146.	540 cm.	Recovery.

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DISCUSSION

DR. JOSEPH RANSOHOFF, Cincinnati, Ohio.—There is a case in the literature reported by a Chicago man, who resected 21 feet of the small intestine; the patient recovered, and he was sued for malpractice.

PRESIDENT HAGGARD.—We have with us today a distinguished guest, who is going to entertain us tomorrow evening, but I am sure the members of the Association would like to hear a few words on this subject from Dr. Martin.

DR. EDWARD MARTIN, Philadelphia.—I have only a word or two to say in connection with Dr. Cannaday's paper. The doctor has shown his resourcefulness in resecting so much of the intestine in these cases, and the promptness with which he did it and remedied the obstruction in a proper surgical manner is commendable. I take off my hat to any man when he has the indications and operates and delivers the goods.

THE CANCER PROBLEM[1]

By Charles H. Mayo, M.D.
Rochester, Minnesota

Cancer continues to be one of the greatest scourges of the human race. It is apparently on the increase among the more highly civilized people, yet may be worse than suspected among lower types of the human race through lack of examination and investigation, as is shown to be the case in cancer of animals and lower types of life. In this connection the work of Erwin Smith is of the utmost interest in showing cancer in plant life. Discussion, investigation and discovery have given control of other great life-destroying diseases and has been most helpful in aiding in the control of cancer. The educational propaganda of Dr. Bloodgood has been of incalculable value in the prevention and early treatment of cancer in this country. The greatest advance in our knowledge of cancer has come from a study of the individual cancer cells. Dr. MacCarty shows the disease to begin in the waiting or repair cells. We thus see how the stimulation of continued traumatism may stimulate to activity such repair cells when lack of control, differentiation and migratory hyperplasia would place them within the cancer classification.

Many writers believe there is a true fertilizing agent of the nucleated cells which gains entrance to local areas and cell intelligence for growth carriers on progressive activity. Some believe this fertilizing agent is water-borne. All of

[1] Abstract by author.

these theories should be subject to investigation, as after all some of the essential facts concerning the cause of cancer are lacking, and yet all causes may be associated and harmonious.

While cancer may be found at any age it is not common under thirty-five and is rare under twenty. The resistant influence of youth may exist in the rapid reduction and restoration to normal of the acidity developed in cell activity, the chemical change being much slower in degenerated tissue or in the normal degeneration of advancing age. We thus have a chemical theory of cancer. Nearly one-third of the cancers that affect man are found where acidity is constant and high as in the stomach. The duodenum which, by right of continuity of tissue, close association with and opportunity for grafting, and apparently far more commonly affected by ulceration than the stomach, is almost never affected by cancer, which, however spreads into all other tissues around it when developed from gastric cancer. Ulcers occur in the stomach one-third as frequently as in the first portion of the duodenum. Sixty to 70 per cent. of gastric cancers give a history of preceding ulcer. Ulcer then is more common in the naturally alkaline duodenum which is periodically bathed with the acids from the stomach, the neutralizing process occurring in the first portion of the duodenum. Ulcer or cancer is most uncommon in the duodenum below the first two inches. Cancer occasionally occurs by penetrating through a duct, although in the whole length of the small intestine occurs but twice to ninety-eight times in the large bowel.

The colon is frequently affected by cancer because here again we have acidity, and 75 per cent. of them locate in the fixed and tissue-surrounded portion of the colon, which retains the dry and harder contents as a traumatic agent. The same statement can be made concerning cancer developing in acid fields—the cervix uteri and urinary bladder, also the mouth—as here cancer usually follows chronic septic

or irritative conditions in which the mouth discharges are acid instead of normally alkaline. The mammary gland, the uterus and the prostate are subject to carcinoma, being tissues in which degeneration is a normal process, having but a limited period of functional activity; the acid field thereby developed makes cancer possible. While ulceration, under proper conditions, may permit cancer to develop, ulceration itself is probably usually bacterial in origin. The stomach is subject to repeated traumatism among civilized people by the daily taking of very hot drinks, which might act to stimulate the repair cells into uncontrolled activity or develop a field for a fertilizing agent.

The essentials of cancer are uncontrolled hyperplasia developed in repair types of nucleated cells and grows most favorably in acid fields.

THE THERAPEUTIC USE OF RADIUM IN GYNECOLOGY

By John G. Clark, M.D.
Philadelphia, Pa.

I purpose to review briefly the results obtained by radiotherapy in two types of gynecological cases: (1) those suffering from profuse or irregular uterine hemorrhages induced by the smaller myomata, and (2) the so-called myopathic hemorrhages of benign origin arising from muscular or vascular changes in uteri of women approaching the climacterium. The second class comprises the cases of inoperable cancer of the cervix, vagina and external generative organs. We have available for study 100 cases of each class, a unique coincidence, which rounds out the two series for easy percentage estimates. To my associate, Dr. F. E. Keene, I am deeply indebted for his careful analyses of the case histories and his accurate tracing of the after-results from which our conclusions have been drawn. Realizing that we were entering a new field, we have endeavored to keep free from an overenthusiastic optimism, an error to which one is liable in viewing the hopeless cancer case, which occasionally shows the most astounding improvement, and to witness the almost invariable relief of hemorrhagic symptoms in benign cases hitherto subjected to hysterectomy.

In presenting a remedy for the relief of symptoms and cure of cases of chronic metritis and small myomata uteri, certain conditions must be fulfilled if it is to serve as a worthy

competitor for myomectomy or hysterectomy. Briefly stated, the remedy must effectively cure, must be attended by as small or be even less hazard than a well-executed operation, and must be unattended by immediate or remote complications. Further, the cure must be permanent. To meet the well-nigh ideal statistics of a well-ordered series of operations in the hands of a skilful surgeon is no small task, for we all have reason to be proud of surgical accomplishments in this type of cases. No one has taken up the use of radium in the treatment of myopathic or myomatous conditions with a spirit of greater conservatism—indeed, with a spirit of skepticism—than we who have had a long series of excellent results from the surgical treatment in this field. We first tentatively experimented with this treatment in cases during their menopausal years, but as our experience has grown we have dropped down the line to younger individuals because of the successful issue in the climacteric cases.

As the question now stands (but we are still in the experimental stage), we only use radium in very light dosage in the treatment of young women in whom a tumor is not palpable but who is suffering from a depleting flow at the periods. The flow diminishes, as a rule, to within normal limits, but occasionally a permanent amenorrhea results. The latter crisis can and will surely be precipitated by too large a dosage. Further, we do not employ and thus far see no valid reason for discarding an operation when the tumor is massive and by its presence is handicapping the function of adjacent organs by pressure. Large tumors are much more likely to be accompanied by inflammatory lesions, and there is always an element of doubt in the diagnosis. In such cases we believe we serve the best professional ends in advising an abdominal operation which permits a complete survey of the entire pathological field and the removal of such tissues as are necessary to restore our patient to health.

In young women in whom a myomectomy is possible we advise operation, for in these cases the uterus may be restored

to normal and the ovaries are preserved. Briefly summarized, we may say that we apply radium in the following group of cases:

1. In large dosage, 50 mg. for twenty-four hours in women over forty years of age suffering with myopathic hemorrhage or menorrhagia from myoma. In such cases we expect to bring on the menopause, and practically all are successful.

2. In smaller dosage, 25 to 50 mg. for from three to eight hours in young women with excessive menses incident to myopathic changes, excessive functional activity of the ovaries in very small myomata and in polypoid endometritis.

We do not employ radium:

1. When the tumors are larger than a three to five months pregnant uterus, or

2. In young women with single myomata which may be removed by myomectomy.

We have now completed a series of 100 cases, all of which are available for statistical study. They are divided as follows:

Myoma	60
Chronic metritis	36
Polypoid endometritis	3
Cervical stump	1
	100

This series of 100 cases comprises patients who have been treated for uterine hemorrhages of myomatous or myopathic origin. In no case was the tumor of large size, and in each the only symptom was excessive bleeding.

Technic of Application. In every case a preliminary curettage was performed and the curettings examined to eliminate the possibility of carcinoma of the fundus. The radium, protected by platinum and rubber, was inserted to the fundus of the uterus, where it remained for from eight to thirty hours. In by far the larger number of cases the time

of application was twenty-four hours and the amount of radium 50 mg. The patients were discharged from the hospital in from three to five days after operation. In the light of our review of cases we are inclined to reduce the dosage to 50 mg. for twelve instead of twenty-four hours, believing that we will serve an equally satisfactory purpose with possibly less menopausal manifestations in some cases.

Results. Nitrous oxide anesthesia is usually employed and the discomfort of the patient is minimal. The shorter stay in the hospital, with practically no lingering sequelæ, is a large factor in hospital economics and favors a quick return to working efficiency on the part of the patient. The postoperative convalescence is so nearly negative as to make it in no way comparable in danger or possible sequelæ to that of even the most ideal abdominal operation. As in the cancer cases, nausea and vomiting followed, lasting one or two days in only 25 and was absent in 58. In a percentage of these cases the preliminary morphinization in the gas administration may have been responsible. Occasionally there was slight elevation of temperature, and in one instance 103° F. was reached. Only very occasionally was the convalescence marked by disagreeable symptoms.

We have obtained successful results in all but 2 cases. Five patients were between seventeen and twenty-five years of age; in 3 of these the profuse menorrhagia has been reduced to a normal flow. Five patients were between seventeen and twenty-five years of age; in 3 of these the profuse menorrhagia has been reduced to the normal flow. There were 5 cases between twenty-five and thirty years of age, 13 between thirty and thirty-five years, 16 between thirty-five and forty, and 61 over forty years. In all except 2 cases the periods have entirely ceased for intervals varying from two to nineteen months. Amenorrhea may immediately follow the application of radium; there may be one or at most two scanty periods, or slight, irregular bleeding may occur for a few weeks before the permanent amenorrhea is

established. In several cases a scanty flow was seen after several months, but this was again followed by amenorrhea.

We have been interested in the action of radium in its effects on leucorrhea. Our results show that in the majority of cases a yellowish discharge follows its application. Rarely a profuse discharge may be present for a few weeks, but is not permanent. Nineteen of our patients gave the history of profuse leucorrhea, and this has been entirely relieved by the radium. In the relief of this symptom we have been greatly surprised, since it seemed logical to assume that the changes produced in the endometrium and uterine blood-vessels might lead to a permanent and troublesome discharge.

Abdominal or pelvic pain has followed in 30 cases, coming on immediately after the application and lasting a few days. It is of interest to note that varying symptoms of the menopause, between slight and severe, have been developed in 53 cases and have been absent in 37.

Only two unsuccessful results occurred: One in a case of chronic pelvic inflammatory disease, in which the patient's chief complaint was excessive and irregular menstruation. This case was diagnosed incorrectly as a myopathic hemorrhage, and there was a lighting up of the old process, since two days after the application the symptoms of acute pelvic peritonitis developed and at operation a few weeks later the findings were those of an old infection with a superadded acute process.

In a second case the radium failed completely after two 50 mg. applications of twenty-four hours, each in the presence of multiple myomata. When the uterus was removed no visible effects of the radium could be detected either macroscopically or microscopically upon the tumors or the ovaries.

Conclusions. Radium is the treatment of choice in all cases of small, uncomplicated myomata whose only symptom is hemorrhage. In these as well as in cases of myopathic hemorrhage almost 100 per cent. cures may be expected. Sufficient dosage will produce complete and permanent

amenorrhea. In young women radium should be used with the greatest caution, since its application may be followed by a premature menopause. If other measures have failed radium may be employed, but in small dosage. Irregular bleeding due to pelvic inflammatory disease, tumors of large size or tumors of any size complicated by disease of the adnexa or surrounding structures, as well as single myomata in young women, are to be treated by appropriate operation and not by the application of radium. In no case of uterine bleeding should radium be used without a preliminary curettage and microscopic examination of the curettings by a competent pathologist. In properly selected cases the immediate convalescence is smooth and restoration to health is complete. Pelvic pain rarely occurs, and, as a rule, is of but a few days' duration. In the exceptional case, leucorrhea follows the application of radium, but this is not permanent. Not infrequently radium has cured or diminished discharge which was present before its application.

Use of Radium in Myopathic Hemorrhage

PATHOLOGIC CONDITIONS

Myoma	60
Chronic metritis	36
Polypoid endometritis	3
Cervical stump	1
	100

AGE OF PATIENTS

17 to 20	2
20 to 25	3
25 to 30	5
30 to 35	13
35 to 40	16
40 to 45	28
45 to 50	23
50 to 55	10
	100

INFLUENCE OF PERIODS

Immediate cessation	40
One period	18
Two or more periods	23
Return to normal and regular	15
No relief	2
Not traced	2

RETURN OF BLEEDING

None	74
To normal after months of cessation	4
Once or twice only then amenorrhea	1
Profuse	1
Not stated	8
Not traced	2

LEUCORRHEA FOLLOWING RADIATION

None	44
Profuse with complete cessation	19
Slight	26
Cured by radium	5
Not stated	6
	100

PAIN FOLLOWING RADIATION

Absent	64
Present	30
Not stated	6
	100

NAUSEA AND VOMITING FOLLOWING RADIATION

None	58
Present	25
Not stated	17
	100

COMPLICATIONS

Present (pyometra)	1
None	97
Subsequent operation necessary	2
	100

SYMPTOMS OF MENOPAUSE PRESENT IN FOLLOWING CASES

17 to 20	1
20 to 25	1
25 to 30	1
30 to 35	8
35 to 40	8
40 to 45	19
45 to 50	10
50 to 55	5
	53
None	37
Not stated	10
	100

Use of Radium in Inoperable Cancer of Cervix, Vagina and Urethra. The lapse of time since we began the use of radium for cancer of the female genito-urinary system does not permit us as yet to regard it as an approved curative remedy, for our period of observation has extended over only three years and the five-year test has not been passed. While therefore we must hold in abeyance any forecast as to ultimate results, our series of 100 cases nevertheless justifies us in estimating with great assurance the excellent palliative effects of radium. Before the advent of radium in the treatment of cancer of the cervix we were constantly passing in clinical review large numbers of cases, among which only the occasional one was amenable to surgical measures; the others, upon being classed as inoperable, dropped into the hopeless discard and frequently did not appear in our card

indexes. A very few only of the latter class ever reached the hospital ward, for beds could not be utilized for this hopeless class of patients. Because of this casual treatment of these patients we did not realize the extraordinarily large disproportion between the operable and inoperable cases, and in former years I offered my small series of radical operations for cervical cancer with a puzzled query, for if cancer was so frequent as was constantly asserted why was not a larger number of operable cases being admitted to our wards: In view of our more recent experience the answer now comes with overwhelming force, and a surgical optimism, never enthusiastic, is now reduced to even a lower degree, for while by the more radical method we operated upon only about 60 cases in ten years, or an average of about 6 annually, we now estimate that we were turning away during the same decade at least 300 as hopeless. A basis for this assertion is laid upon the statement that during the last three years we have refused no patient regardless of the extent of the growth, giving all the possible benefits of radium, 100 having been admitted to the hospital. Combined operative statistics show that even in the selected cases for a radical hysterectomy the primary surgical mortality will seldom fall below 10 per cent., that the disabling sequelæ are numerous and serious, and that in the end, under average skill, at least 66⅔ per cent. will die from a recurrence. We have, therefore no reason to boast of epoch-making strides in the surgery of cancer of the uterus. Because this minimum salvage is the best that has been obtained by any other means of treatment, we still strongly adhere to the dictum that when the case is within surgical bounds it should be treated by radical surgical measures.

However, the remarkable palliative results in cases of inoperable cancer of the cervix have modified our view-point as to the cases which fall within the elective surgical class, for our former rule was when in doubt as to the extent of the growth, operate, but now we reverse the policy and in doubt-

ful cases we employ radium. Under this restrictive policy our percentage of operative cases has shrunken, but we trust that the results from a radical hysterectomy will yield better curative results. We justify this conservative attitude by the assertion that in 100 inoperable cases treated by radium local healing has occurred in 52 per cent. This does not mean, however, that all of these 52 patients are alive, for several are dead of metastasis but without return of the local cervical process primarily destroyed by the radium. Even though the patient died from the secondary invasion this is a remarkable showing as to palliative results, exceeding in our experience all other remedies, and we trust that several will survive the five-year test.

One of the terrifying symptoms for the patient and her family is hemorrhages. Of our 100 cases 60 were completely relieved of this symptom. There was a recurrence of hemorrhage in 10 cases, and in only 4 was it uninfluenced. Vaginal discharge other than hemorrhage was checked in 51 and uninfluenced in 15. Of those suffering pain 23 were relieved and 14 were not relieved. In some cases the relief of pain was only temporary, varying from a few days to a month or more.

All statistical data concerning our study of these cases appear in the appended tables.

A few surgeons have advocated the use of radium preliminary to a hysterectomy. From this view we are radical dissenters, for it hardly appears conceivable that a remedy which works effectively for at least 2 cm. from its point of application can be improved upon by a surgeon's knife, limited by such insuperable anatomical barriers as the bladder in front, the rectum posteriorly and the ureters and pelvic vessels laterally. We therefore adhere strictly to one rule, namely, never to attempt an operation on any case that has been healed locally by radium. It appears to us a most unwise surgical policy to subject a patient to the hazards of a radical operation, rendered more dangerous by the dense

cicatricial tissue which forms in the wake of a successful radium application in what we believe to be a fatuous attempt to secure still more effective results. Indeed, we are inclined to the view that nests of degenerating cancer cells may actually be liberated and thus render abortive an otherwise good palliative result.

Radium, as is shown in our series of cases, is by no means an infallible panacea for cancer. There is no way of determining which cases will be benefited by its use. There is beyond doubt a certain percentage, as estimated by our observation, in which cancerous growths are not retarded by radiotherapy; indeed, in an occasional case it would appear that there is a positive acceleration of the growth. That many cases show an astounding improvement, with local disappearance of the ulceration, cannot be gainsaid. In our series several instances occurred in which the results achieved were so remarkable as to be almost incredible. In some of these cases the fire is but smouldering and will break out sooner or later with renewed violence, as several of our cases have proved; but upon the whole this treatment yields, to say the least, highly encouraging palliative results, and we trust that permanent results will follow in a definite percentage of otherwise hopeless cases.

In the discussion of cancer cases the question of hospital economics must not be lost sight of. The length of stay in the hospital following a radical operation will average at least three weeks, whereas after the application of radium not more than three days will be required. When complications arise after radical operations they are usually serious, entailing much suffering. Such patients on returning home are likely to remain semi-invalids for several weeks or months, and when, as is frequently the case, there is a rapid continuation of the disease they become a heavy burden to a poor family. In the comparison of statistics this difference between the two classes of patients is greatly in favor of those treated by radium, a fact that tends to incline us

strongly to the use of radium in border-line cases, which we formerly subjected to a radical operation.

Removal of the uterus in cases of cancer of the fundus has yielded such good results that I do not feel we are justified in taking any chances with radium, even in the border-line cases, except in cases in which there is a serious surgical contra-indication. In our series of 100 cases only 4 were thus treated. *Our surgical attitude toward the cervical and fundal growths is diametrically opposite. In border-line cases of cancer of the cervix we invariably employ radium. In advanced cases of cancer of the fundus we invariably perform a hysterectomy.* A pessimistic view dominates our outlook in the surgical treatment of the cervical growths of the pathological process is at all advanced, whereas fundal growths may be viewed with a cheerful optimism even when the cancerous process is extensive. As a palliative agent we may assert, with full assurance, that we have never obtained results with any other method that have even approached in beneficence those secured by radium. The treatment is simple and entails no distress to the patient, and the results in checking hemorrhages are immediate and in a very large number of cases never returns even in the fatal cases. Because of the well-nigh miraculous action of radium in the occasional case there is danger of unbridled optimism, and no one should let the occasional astounding result cloud his vision when it comes to a judicious consideration of a large series of cases.

In this brief summary we have attempted to give a fair and unbiased statement of the benefits, limitations and dangers of radiotherapy in gynecology. Our conclusions of today may be completely changed by our experience of tomorrow, for the entire field of work is still in the period of probation; but we are certain that the benefits of this remedy are so great that no well-ordered clinic can do the best work without it. In our experience 100 mg. have been sufficient to serve a most satisfactory purpose in gynecologic practice.

Use of Radium in Inoperable Cancer of the Cervix, Vagina and Urethra

DIAGNOSIS

Carcinoma of the cervix	74
Carcinoma of the fundus	4
Carcinoma of the vagina	7
Recurrent	10
Urethra	3
Chorio-epithelioma	2
	100

AGE OF PATIENTS

20 to 30	4
30 to 35	2
35 to 40	6
40 to 45	11
45 to 50	17
50 to 55	22
55 to 60	13
60 to 70	19
70 to 80	6
	100

History of Patient after Application of Radium

HEMORRHAGE

Complete relief	60
Recurrence	10
Uninfluenced	4
Not stated	15

LEUCORRHEA

Checked	51
Uninfluenced	15
Not stated	23

PAIN

Relieved	23
Not relieved	14
Not stated	52

FISTULÆ

Rectovaginal	4
Vesicovaginal	4
Local healing	52
No effect on growth	8
Hastened growth	2
Immediate untoward result	0
Stay in hospital, average three days	
Patients not traced	2
Too recent to report	9

Results following Radium Treatment

DIED

2 to 4 months	7
4 to 6 months	6
6 to 8 months	7
8 to 10 months	5
10 to 12 months	0
12 to 14 months	3
14 to 18 months	3
18 to 22 months	0
22 to 26 months	1
26 to 28 months	0
28 to 30 months	0
	32

ALIVE

2 to 4 months	7
4 to 6 months	8
6 to 8 months	6
8 to 10 months	6
10 to 12 months	9
12 to 14 months	6
14 to 18 months	6
18 to 22 months	2
22 to 26 months	1
26 to 28 months	1
28 to 30 months	3
	55

Died a few days after leaving hospital from heart failure	1
Died (time not stated)	1
Not traced	2
Too recent to report	9

DISCUSSION

Dr. C. Jeff. Miller, New Orleans, Louisiana.—I am particularly interested in the statistics related by Dr. Clark, as I have been using radium for about the same length of time and in practically the same dosage recommended by him. We practically agree as to the indications for radium and I am gratified to learn that our results are almost identical.

I have not been using as large dosage in controlling bleeding due to the metropathies and small fibroids as some of the authorities, and find that the results are just as satisfactory. I found that a smaller amount of radium was sufficient and that more careful screening minimized the amount of pain frequently complained of when a larger dosage was employed. I should like to ask Dr. Clark if he continues to screen only with rubber tubing? I used celluloid capsules without rubber and found that the reaction was often attended with soreness and colicky pains that often continued many weeks. I have had occasion to examine the interior of the uterus in two instances in which the uterus was removed subsequent to radium applications. The cavity of the uterus was almost obliterated and a small greenish slough was present in both cases. I am confident that insufficient screening had been employed in these cases.

The value of radium in controlling uterine hemorrhage and even the menses when necessary is established beyond a question of a doubt. Whether or not it will be possible to so regulate the dosage that the menstrual flow may be reduced without checking it, and at the same time produce a minimum of permanent changes in the tissues, is yet undetermined. I have been able to reduce excessive menstruation in four young women by short exposures repeated after two months. They are perfectly well so far as the menorrhagia is concerned, but whether or not their procreative powers have been destroyed remains to be seen.

I had occasion to review my results in carcinoma of the uterus some months ago and found that my statistics tally with those of Dr. Clark

In regard to the danger of fistulæ after radium treatment, I will state that it has occurred in five cases of my series. It cannot be said, however, that all of the five were due to radium. We must not forget that fistulæ develop in the course of advanced carcinoma when radium is not used. I believe that careful screening of the vaginal wall, especially the posterior wall, will

minimize this complication. All of the above fistulæ were in the rectovaginal septum.

In conclusion, I wish to emphasize everything Dr. Clark has said as to the value of radium in small fibroids, hyperplastic conditions of the uterus and uncontrollable bleeding of the menopause. Radium acts so satisfactorily in such cases that it may be said that it is almost sinful to resort to radical surgical measures when radium is available.

Dr. Guy Le Roy Hunner, Baltimore, Maryland.—I have not reached that state of opulence where I can afford to buy radium, so my experience with these cases is simply one of sending them to someone else that commands radium. I have had very satisfactory experience in sending several cases of recurrent carcinoma, where the growth returned locally in the vagina, to Drs. Kelly and Burnam, and I am now following these cases for two or three years after radium treatment, and there has been as yet no evidence of recurrence. It seems to me that if radium were to be used in this one class of cases alone it would be a tremendous therapeutic measure. With reference to those cases which Dr. Miller and Dr. Clark have spoken of, young women in whom, when we curette the uterus, we find hyperplasia of the mucosa, and perhaps on examination we find they have the fibrocystic type of ovaries; if radium is of value in that one class of cases, that bleed and are so anemic, and in which we have such unsatisfactory results from repeated curettages, it is an immense therapeutic asset for them.

I have reached the stage in which I do not feel justified in operating on these comparatively small fibroids, and unless these patients have some other intra-abdominal lesion, such as appendix trouble, or evidence of some such trouble that needs an abdominal operation, I refer them to the radium institute.

It seems to me, one great lesson we can draw from the reports Drs. Clark and Miller have given us is one of humility. We can all remember in a very recent past period when there were acrimonious attacks on Dr. Kelly for his earlier reports on the use of radium. We all know the enthusiasm that Dr. Kelly displays with every new subject he attacks, and perhaps there was some ground for criticism, but now when we see these men making such favorable reports as we have heard today, it certainly should be a lesson to us to be a little careful about condemning the man who is bringing forward some new point in medicine and wait until he gets further results before drawing a final conclusion.

Dr. Joseph Ransohoff, Cincinnati, Ohio.—When Dr. Clark closes the discussion I would like to ask him to tell us why he curettes cases of fibroids of the uterus before he uses radium? We have treated a great many cases of fibroid tumors of the uterus with radium and, as a rule, without any previous curetting. I would like to get some reason why curetting is necessary before the use of radium.

In one of the very first cases in which we used radium five years have elapsed since the radium treatment was begun. It was a case of carcinoma of the uterus in a woman of fifty, who absolutely refused operation. A section of the tissue removed was proved pathologically to be carcinoma, and that woman is well today.

Dr. Clark tells us that there are some cases in which he refuses to use radium. In thinking over my cases I do not recall one that I have refused to treat with radium. In some of these cases that seem almost hopeless we sometimes get the best results. One such case was brought from Charleston not long ago on a stretcher ready to die, but in a short time, after two or three applications of radium, the woman began to improve and is now clinically well, so that relatively few cases of carcinoma of the uterus should be considered so hopeless that we should not try the use of radium.

In regard to operative procedures on elderly women, if I understood Dr. Clark correctly, he operates on younger women with small fibroids. In that class of cases I have found that radium is peculiarly applicable because these women do not want to sacrifice their uteri. I have seen some enormous fibroid tumors disappear after the use of radium in which operation would have been fatal. In a series of cases I recall one patient with an enormous fibroid which extended almost up to the xiphoid cartilage who could not be operated upon because of an aneurysm of the aorta. That fibroid disappeared under the use of radium, and that woman died three years later from rupture of the aortic aneurysm.

The most satisfactory results are those obtained from radium in carcinoma of the cervix, with fetid discharges, with a lot of bleeding. I can assure you that 60 to 70 per cent. of these women are going to be benefited by radium in a way that they cannot be benefited by any method of procedure at our command. The discharge stops during the course of a week or ten days, and, as a rule, not to reappear. These women improve from the first application of the radium in a way sometimes that is really surprising.

The most difficult cases we have encountered are those in which operation has been done and in which the scar nodules recur. I refer particularly to those cases in which the nodule occurs in the posterior cul-de-sac. Those are the cases that give us the most concern because they can only be treated through the rectum.

Since we are on the subject of the value of radium, I would say that one of my personal friends, a physician, was taking two grains of morphin every day for carcinoma of the prostate. After a single application of 100 mg. of radium to the rectum, in twelve hours the pain disappeared. The growth has decreased in size about three-quarters, as recognized by a genito-urinary specialist, and the man from the time of the first application of the radium has not taken any more morphin. It relieves pain if it does that. Furthermore he gets along now without catheterization.

Dr. J. Shelton Horsley, Richmond, Virginia.—I should like to ask Dr. Clark as to the proportion of cases of adenocarcinoma of the cervix.

Dr. I. S. Stone, Washington, D. C.—I should like to ask Dr. Clark as to the effect of radium on the internal secretions in young women. I feel very much impressed with his excellent exposition of the subject and the results he has reported. If radium does have an effect on the internal secretions of an untoward character I would like to know it.

I have seen peculiar and undesirable results from the use of the *x*-rays in more than one instance. I had a case of that kind brought to my notice recently. From the disclosures that have been made, there is much to be learned about the use of and effects of radium, and considering the mere fact that these gentlemen are so careful not to give too much radium, what does that portend? I am seeking this information because in all humility I know nothing about radium myself; I send my patients to those who use it. I have seen the beneficial results of it in many instances. I am obliged to ask that question because my experience does not satisfy me that its use is devoid of dangers. Does it, like the *x*-rays, produce undesirable symptoms?

Dr. James K. Simpson, Jacksonville, Florida.—I should like to ask Dr. Clark what percentage of cases in which radium is

used in treating fibroids in young women show either cessation of the growth of the fibroid, actual atrophy or disappearance of the tumor following its use? Also, whether this class of cases has been studied pathologically following the use of radium to determine if there is any effect upon the bloodvessels such as an obliterating endarteritis or phlebitis?

Dr. Clark (closing).—I feel very much as Dr. Miller does relative to the effect of radium on the ovaries. I have observed these cases with considerable interest, and what I am about to say concerning its effects on ovarian activity will probably answer Dr. Stone's question. A small dosage of radium (50 mg.) applied from three to six hours in younger individuals does not seriously disturb the patient and menstruation tends gradually to return to normal, with no untoward symptoms, which may be ascribed to disordered internal secretions. I have seen none of the unhappy experiences from the use of radium that Dr. Stone speaks of as a sequel of the therapeutic application of *x*-rays. Radium in proper dosage is practically free from any immediate disagreeable postoperative sequelæ. We have used 50 mg. as a dose in these cases, and in twenty-four to forty-eight hours thereafter there was cessation of pain the same as after a simple curettage. I coincide therefore with Dr. Miller in the belief that judiciously administered the fear of serious effects of radium on the ovaries has possibly been exaggerated, for I too am inclined to the view that the radiant energy is expended chiefly on the uterus.

In reply to the question of Dr. Simpson, it is asserted by other observers that on macroscopic examination arteriosclerosis of the uterine vessels differing little from that found in all uteri of women beyond thirty-five years of age is observed, and it is difficult to say that the sclerosis produced by the radium is any more intense.

As to the method of application, I have had the same experience as Dr. Miller. We use a glass capsule which is contained in a platinum cartridge; this in turn is enveloped in black-rubber tubing. I am indebted to Dr. Miller for his suggestion of cutting down the number of radium hours from twenty-four to twelve hours in the usual myoma case.

As to Dr. Ransohoff's question as to why I curette before using radium, I will say that the curetting is done for diagnostic purposes. We want to be sure that we are not applying the radium

in a carcinoma case under the mistaken diagnosis of myoma uteri. In other words, if we have 100 fibroid cases we want to be sure that there are not mixed with them three or four or possibly more cases of carcinoma of the fundus which we have overlooked. As a basic principle, therefore, we examine the curetted specimen in every suspected case. If I could feel sure that I was dealing with a simple fibroid case I would not curette the uterus because the therapeutic issue is not enhanced and there is, of course, a small element of greater risk connected with it.

As to the inoperable cases of carcinoma, we have not as yet turned any away, but I have been thinking lately we would, as we now know that we cannot save or greatly ameliorate the symptoms in the cases very near a lethal terminus. However, since the use of radium does not do these cases any particular harm, I appreciate Dr. Ransohoff's position in this regard.

As to the remarks of Dr. Horsley concerning the type of carcinoma, I cannot give the percentage of cases of adenocarcinoma in our series, but so far as our experience goes, we see no difference between adenocarcinoma and the epithelial type so far as the action of radium is concerned. We apply radium regardless of the type and one apparently reacts about the same as the other.

As to Dr. Simpson's question relative to the decrease in size of the myomatous tumors, I will say yes, we do see a decrease, but it may take six months before it is appreciable. The symptoms for which we apply radium disappear long before the tumor decreases in size. I cannot answer the question from personal observation as to the direct effect of radium on the arteries and veins.

In regard to what Dr. Hunner has said about not being rich enough to afford this therapeutic luxury, I do not believe that any one of us needs to spend a large amount of money to secure radium. In any city a group of active surgeons can form a syndicate and secure say $6000 or $10,000 worth of radium and each can make use of it on an equitable basis without undue burden to any one individual. I have radium in my department at the University Hospital which is just as accessible to poor patients as to the well-to-do. I am also in happy accord with Dr. Hunner in his tribute to Dr. Kelly. Through his faith in this remedy, backed by a very heavy financial outlay, he has established the principles of treatment in this country and has advanced the issue quickly to a splendid therapeutic epoch.

To Dr. Kelly, therefore, is due all credit as the originator of the radium idea in America.

In conclusion, I had a rather amusing experience with one of my Jewish doctor friends who brought for treatment a patient with an inoperable carcinoma of the uterus. The husband was rather apathetic and showed little interest in our conversation, until I told the doctor that I thought this was a case in which probably radium would be of some help. I said that I would use 100 mg. of radium, the cost of which was $10,000. When the husband realized that such an expensive remedy was to be employed at a very small cost for the treatment, the lovelight flashed from his eyes and he said, "Vell, Rachel, $10,000 vorth of medicine for twenty-four hours ought to do you good."

TWO UNUSUAL CASES OF MALIGNANT TUMOR: (1) MALIGNANT TUMOR OF THE TESTICLE IN AN INFANT, (2) MELANOTIC SARCOMA OR MELANOMA OF THE SUPERIOR MAXILLA

By William B. Coley, M.D.
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Case I.—R. H., male, aged twenty months, was referred to me by Dr. D. P. Murphy, of Elmira, N. Y., with the following history: The patient had always been well until May, 1917, when the mother noticed a swelling of the left testicle—there had been a slight injury to the testicle by striking it against the side of the baby-carriage a few months before. The swelling increased very rapidly, and at the end of three weeks had reached the size of an orange. The skin was of a purplish hue and covered with many dilated veins. Three weeks after, the testicle was removed by Dr. Murphy and the wound closed. At the end of three weeks a local recurrence took place and the tumor grew with great rapidity, until in a few weeks it had reached the original size. A second but ineffectual attempt was made to remove the growth. This was followed almost immediately by a second recurrence, which grew with great rapidity, and became fungating over the distal portion; no enlarged glands were observed.

Physical Examination. The physical examination at the time of his admission to the Memorial Hospital (October

19, 1917) showed a large tumor, elliptical in shape, about the size of two fists, occupying the entire left scrotum, the distal portion being greater in circumference than the proximal. The distal end was covered by a fungating mass, with an extremely foul discharge. The mass was freely movable and did not extend up into the inguinal region; the cord was apparently not involved. At the time of the first operation the clinical diagnosis was "hydrocele" and the pathological report "non-malignant."

On the same day he entered the hospital, under ether anesthesia I removed the entire tumor as far as the external ring. The cord was not enlarged nor apparently involved. Enough healthy skin from the other side of the scrotum was found to close the wound. Pathological report by Dr. James Ewing (December, 1917): "The specimen is a round, solid, soft elastic tumor mass, 7 x 8 cm.; it fungates through the skin over an area 4 cm. wide, a portion of skin accompanying the specimen. On section the tumor is smooth, translucent, hemorrhagic along the fungating edge. It is circumscribed by an indistinct capsule. No portions of the testis are visible in the single gross section. On microscopic examination the structure is composed of large and small groups of large polyhedral and spindle cells of indifferent embryonal type, consisting chiefly of hyperchromatic nuclei. These cell groups grade off into an abundant mucinous tissue of myxomatous type and every gradation from polyhedral to star-shaped cells may be followed. Some cell clusters surround bloodvessels, which are not numerous. There are scanty small points of necrosis. The diagnosis is embryonal carcinoma of testis, with transition to pseudosarcomatous structure."

The patient was immediately put upon the mixed toxins of erysipelas and Bacillus prodigiosus, beginning with a dose of $\frac{1}{10}$ minim and increasing by $\frac{1}{10}$ and later by $\frac{1}{8}$ minim, up to the point of producing a marked reaction, temperature of 103° to 104°. The wound healed promptly, with the

exception of the site of the drainage tubes, and at the end of a month he returned home, where the toxins were kept up by the family physician, Dr. Murphy. At the present time, June, 1918, there has been no recurrence, and the patient is in good health.

In view of the prompt, almost immediate recurrence after the first two operations and the failure to recur after the third it would seem fair to infer that the toxins had some inhibitory influence in preventing a return of the disease. Of course, it is too early to make any prognosis as regards the final result.

CASE II.—*Melanotic sarcoma or melanoma of the superior maxilla.* Melanotic sarcoma, as it is well known, is chiefly primary in the skin and in the choroid of the eye. A few cases have been reported in which it originated in the matrix of the nail and in the region of the anus, but there are very few cases on record in which it started in the mucous membrane.

Bland Sutton in his book on *Tumors, Innocent and Malignant,* states: "Primary melanoma of the mucous membrane is very rare; and it is odd that the recorded cases have been observed on the mucoperiosteum of the hard palate. Treves, in recording an example, reminds us of the fact that the mucous membrane in this situation is charged with pigment in certain mammals. This is the case with dogs."

Bland Sutton reports one case of melanotic sarcoma originating in the mucous membrane of the superior maxilla. To this small group may be added the following case, which came under the writer's observation:

H. S., male, aged fifty-eight years, was first seen by Dr. Joseph Flint, professor of surgery in New Haven, Conn., in May, 1911, and gave the following history: He had always been well until the spring of 1911, when shortly after the extraction of two teeth in the left superior maxilla a tumor appeared at the same site, which at first grew slowly and then more rapidly, until February, 1912, when the first

FIG. 1.—Case I. Embryonal carcinoma of the testis. Recurrent in a child, aged twenty months.

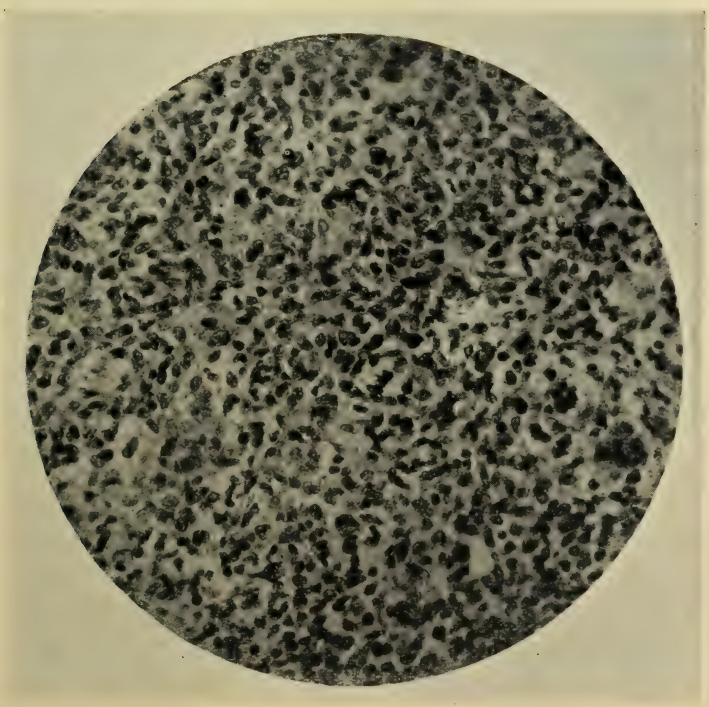

FIG. 2.—Case I. Embryonal carcinoma of the testis in a child, aged twenty months.

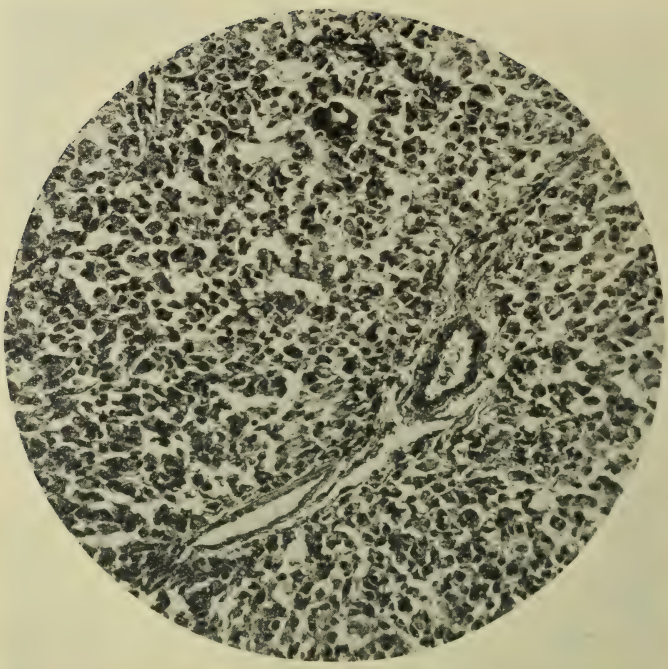

Fig. 3.—Case II. Primary melanotic sarcoma of the superior maxilla.

operation was performed by Dr. Flint. The latter's report states: "I did a partial resection of the superior maxilla. Owing to the rapidity with which the growth was spreading, I later advised him to have a radical operation for the glands of the neck, which, however, he refused. He has just returned to me with an inoperable recurrence."

Microscopic report by Dr. Butler (pathologist of the New Haven Hospital): "The section shows a moderately cellular tumor involving the epithelial layer of the alveolus and the underlying tissue. It is composed of long spindle cells, closely packed together, lying in dense intercellular substance. The epithelial layer is frequently invaded from below by tumor cells, and at one point there is ulceration. Heavily pigmented cells are present below and within the epithelial layer, in foci throughout the tumor, and especially in large spindle and polyhedral cells about the edge of the tumor-cell groups. Diagnosis: melanoma."

The glands of the neck continued to increase in size, and a few months later a local recurrence took place. The patient was referred to me on December 8, 1916, at which time, physical examination showed a tumor extending from the tip of the external ear on the left side, nearly to the upper border of the thyroid cartilage, a little beyond the angle of the jaw and almost to the angle of the mouth. Posteriorly it extended as far as the sternomastoid muscle. The tumor protruded two and a half inches beyond the normal surface of the face, was irregular and globular in outline and apparently attached to the jaw; it was firm in consistence and fairly movable; some parts were softer than others. The skin was normal in appearance except slightly reddened over the most protuberant part. Intrabuccal examination showed in the left side of the hard palate, near the alveolar process, an opening one inch long and a half inch wide leading into the nasal cavity. On the outer and anterior surface of the opening was a black area which appeared to be melanin. This black discoloration led over

the alveolar process to the mucous membrane of the cheek and to the inner side of the lower lip. The teeth on the left side of the upper jaw were lacking; the tonsils appeared normal.

On December 15, 1916, under ether anesthesia, I made an attempt to remove the large tumor of the neck by operation. The external carotid was tied and after extensive dissection the tumor was almost completely removed, although it was necessary to leave some tissue for a flap over the deep vessels. On December 31, 1916, about two weeks after the operation, the mixed toxins were begun and continued up to the point of producing moderate reactions.

On January 3, 1917, 900 mc. of radium emanations were applied (3 mm. lead filter, 3 cm. distance) to the left side of the neck for ten hours.

On January 4 the patient was discharged from the hospital but was later readmitted on February 7, 1917, at which time no change in the condition could be noticed. 96 mc. of radium emanations (platinum and rubber) were applied to the antrum for two hours.

Neither the toxins nor the radium apparently had any effect in checking the growth of the tumor. He continued to fail in general health and the tumor increased rapidly in size, finally proving fatal on May 22, 1917.

This case is interesting in the first place because of its extreme rarity and in the second place by reason of the comparatively long duration of the disease before it began to affect his general health, nearly five years intervening between the first and second operations. The total duration of the disease was six years.

THE ETIOLOGY OF URETERAL CALCULUS

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(From the Gynecological Department of the Johns Hopkins University and Hospital.)

Most of you know to your discomfort that I have been particularly interested in the past two years in the subject of ureteral stricture. I risk becoming a bore and keep hammering away at the subject because I am almost daily learning some new feature about it and becoming more impressed with its importance. I am simply amazed that we have all been overlooking this lesion so long and as thoroughly as we have. As patient after patient consults me, suffering with ureteral stricture and its effects, I ask myself what we have been doing with these patients in the past.

Up to November, 1915, I had recognized 50 cases as suffering with ureteral stricture. At our White Sulphur Springs meeting in December, 1916, I reported on 92 cases, 42 of them having occurred within the year after looking up the records of my original 50 cases and getting especially interested in the subject. In this past year I have seen approximately 200 additional cases.

This experience with about 300 cases of ureteral stricture has been answering in a degree the question I have just mentioned. Their histories demonstrate what has been their treatment in an epoch in which we have failed to recognize the frequency and importance of ureteral stricture as a

clinical entity. Many of them have been fortunate enough to escape operation, and because we could discover no lesion to account for their persistent symptoms, we have put many of them down as neurasthenics, and if they could afford it, they have had rest cures; or because of their gastro-intestinal symptoms, we have sent them to the gastro-enterologists to be drugged without relief, or we have sent them to the orthopedist to have braces for their hip, back and sciatic symptoms.

Of those who have not been so fortunate and who have fallen into our surgical hands the largest number have lost a normal appendix; many have had an ovary removed, some have had hysterectomies without relief. Some have had a useless gall-bladder operation. Many have had a fixation of the kidney without relief of symptoms because the symptoms, the slight hydronephrosis and the prolapse of the kidney all depended on a ureteral stricture. In some the stricture has been neglected so long that we have had to sacrifice the kidney.

This appeals to some of you as an imaginary list of ill-advised treatment, but I can assure you that all of these operations and many more have been recorded in these patients' histories, and some of the victims have not escaped with but one of these operations, but have had two, three or more of them, my most unfortunate patient having had eight operations, seven of them abdominal. These patients are consistent in still complaining of their original symptoms until the ureteral stricture is diagnosed and dilated.

Too many of these cases have been in my own list of useless operations, and I am now consulted frequently by some former patient who tells me of the operation I did one, two or more years ago without giving relief, and brings the typical history of ureteral stricture, which I find in her original records.

History of the Theories Concerning the Etiology of Urinary Calculus. One of the interesting develop-

ments in this study of ureteral stricture and its influence on other important lesions of the urinary tract has been the demonstration that many stones in the urinary tract are probably first formed in the site of a stricture. A review of the literature shows that ureteral calculi have generally been ascribed a renal origin and that the development of ideas concerning the etiology of urinary stone may be classified roughly into three periods. The first period dates from early medical history to the latter part of the nineteenth century, and may be called the "chemical" epoch, in which writers sought to explain the origin of stone in some obscure change in the composition of the urine. The second period includes the last two decades of the last century and the first few years of the present, and may be called the "bacteriological" epoch, in which writers emphasized the influence of microörganisms on stone formation. The third period, overlapping on the second, is synchronous with the recently developing accurate methods of study and treatment of ureteral diseases, and may be called the "mechanical" epoch, in which injuries and inflammatory diseases within and outside of the ureter have been invoked as frequent causative agents in ureteral stone formation.

The chemical theories include those dealing with heredity, race, climate, diet, drinking water, ingestion of alcohol, incidence of gout and rheumatism, faulty metabolism, etc., and date back at least to Galen,[1] who claimed a relationship between stone formation and gout. Paracelsus spoke of an animal-cementing substance from the food, a tartar filled mucus, as essential in the formation of the "sperma calculi." Sydenham and Boerhaave considered a specific diathesis, a characteristic variation of the metabolic assimilation, to be fundamental and to result in a crystallization of the stone-forming substance held in excess in the urine.

Van Helmont attributed stone formation to the coagulation of uric acid, which came from the existence of potential alcohol and the breaking up of urinary ferments.

Fourcroy and Vauquelin considered the organic cementing material to be albumin and gelatin, and they, and later Scheele (1776), investigated the solid elements concerned in stone formation. Scheele referred to the "Steinsaure," the stone acid or uric acid, phosphorus and saccharic (oxalic) acid, ammonia and the alkali earths, and Fourcroy classified the stones as consisting of one, two or more than two of the stone-forming materials.

At the end of the eighteenth century many authors, notably Wilson, 1795, wrote of the influence on stone formation of the frequent use of acid foods and drinks, of the drinking of water rich in lime and of the influence of sedentary habits.

In 1852 v. Walther defined the spontaneous formation of calculi (*i. e.*, those formed without the aid of foreign bodies) as an organically vital process, pointing out that with the same materials outside the living body only a sediment forms.

Meckel von Hemsbach, 1856, advanced the theory that the organic cementing material was the product of an advancing inflammatory condition, a catarrhal affection of the urinary tract, "Meckel's stone-forming catarrh."

In 1884 Ebstein, by careful dissection and analysis of stones, concluded with Fourcroy and Vauquelin that stones formed in the urinary tract contain a framework of albuminous substance. He thought this organic material resulted from a catarrh of the urinary tract and a sloughing of the epithelium, thus forming a groundwork for the impregnation of the inorganic particles of the urine.

Posner accepted these two factors as important, but thought that, in addition to the sloughing of the albumin framework and its impregnation by crystalline bodies, we must have a third factor in stone formation, viz., a slowing of the urinary stream.

Küster[2] reviews the history of the etiology of urinary calculi. He dismisses the views formerly held as to the roles played by climate, drinking water and racial tendency.

Many cases even in the third and fourth decades of life may be traced to the uric-acid infarct in the young. Gout as a cause cannot be proved, although the two conditions are often present in the same individual. He quotes Ackerman as viewing stone formation as due to a retained hyaline cylinder in the kidney pelvis and v. Recklinghausen as considering a blood-clot the foundation for stone. Küster says that if it be true, as stated by Moritz and Mendelsohn, that each stone particle has an albuminous center, then stone formation loses its specific character and belongs on the borderline between a physiological and pathological occurrence.

A. E. Roberts, of the English Army,[3] attempts to explain, on dietetic grounds, the unusual prevalence of bladder stone in certain parts of India where the inhabitants are vegetarians and where gout is almost unknown. Of 3041 cases of stone in the bladder operated in one year in India, 1482, or nearly one-half, were in the Punjab, where the inhabitants eat cereals and leguminosæ as opposed to the rice diet of other portions of India. These diet stuffs "yield urine which is as acid as that due to a meat diet, under normal conditions of digestion, because they are rich in albumin and phosphates and in calculus."

The inhabitants of the Punjab have a deficiency of salt, which is so necessary with a vegetable diet, and Roberts considers this the cause of their prolific calculus formation. He then takes up what Hirsch, in his great work on *Geographical and Historical Pathology*, calls "the mystery of the excessive prevalence of stone in the tropics," while "gout" is practically unknown, and attempts to show that absence of sufficient NaCl and excess of K in the diet renders the blood relatively alkaline, resulting in the dissolving out and excretion of the uric acid as far as the bladder. On the other hand a free ingestion of NaCl results in relatively acid blood, with a storage of the uric acid in the spleen and joints (gout).

The bacteriological theories concerning stone formation

are a natural outcome of the development of bacteriology and a clearer knowledge of the role of microörganisms in pathological processes.

Naunyn's work in showing the relationship between gall-stone formation and infections undoubtedly stimulated similar investigations on the etiology of urinary calculi.

M. L. Harris[4] advanced the thesis that all renal calculi are of bacterial origin, and supported this by evidence collected from the literature and by experimental and clinical data.

"The subdivision of kidney stones by Albarran into primary and secondary stones, the former being considered of non-bacterial origin and the latter of bacterial origin, however, is of great value, as will be seen later, if we base the classification not on the character of the stone but on the state of the kidney. In primary stones the bacteria are eliminated by the healthy kidney. They develop in the urine in the tubules, calices or pelvis, where they lead to the stone formation, as above described, without invading or setting up pathological changes in the kidney proper.

"In secondary stones the kidney is already the seat of active bacterial invasion and the stone formation is subsequent thereto. The great danger of primary stones is that they may determine bacterial invasion of the kidney, in which case they thereafter partake of the nature of secondary stones."

T. R. Brown[5] made accurate bacteriological studies on 7 cases in which infection of the renal pelvis or the substance of the kidney was associated with the presence of stone. Careful qualitative chemical analyses of the calculi were also made, and his conclusions were that with the proteus vulgaris and other alkaline-producing infections we may expect to find stones made up of the alkaline earths, while with the colon and other infections associated with acid urine we may expect to find stones made up of the organic materials such as urates and oxalates.

We have seen that the formation of stone in the urinary

tract has been a subject of intense interest and speculation since early medical history, and that we still have problems to solve concerning its etiology. Scientific knowledge of the diagnosis and treatment of urinary calculus has awaited the development of abdominal and pelvic surgery during the last quarter of the nineteenth century and the early years of this century. This knowledge and accuracy of treatment has been tremendously accelerated by the development of the specialty of urology with its use of the cystoscope, the renal catheter and roentgen-ray photography.

We cannot emphasize the rapidity of this evolution more forcibly than by quoting from a paper by Henry Morris,[6] of London, published in 1884, enunciating the principles concerned in the treatment of calculus impacted in the ureter: "As regards the question of surgical operation in which there is impaction in the ureter, there is no doubt but that in some of them the calculus could be removed by nephrolithotomy or pyelonephrotomy . . . and there is good reason to believe that with the more frequent resort to digital exploration of the kidney, through a lumbar incision, a calculus impacted in the upper end of the ureter will not infrequently be detected, and extracted through the loin. . . . Calculi impacted in the intermediate parts of the ureter are practically beyond the reach of the surgeon."

It has been the development of abdominal and pelvic surgery, and especially of urology, that has led to ureteral surgery and to a gradual appreciation of the importance of mechanical factors in the etiology of ureteral stone formation. A few quotations from recent literature will show that the ideas held by the author are at variance with many of our past ideas, but that they are confirmatory and elaborative of some of the recent theories rather than original.

Israel (1902—"Nierenkrankheiten") says: "The cause of stone formation is still in the dark. One theory is that uric and oxalic stones are due to a poor oxidation of the

proteids. It seems certain that heredity plays a part in the predisposition to stone formation. The uric acid and cystin stones run in families. It is also certain that in some families gout, rheumatism, diabetes mellitus, nephrolithiasis and bladder stones alternate. Subcutaneous trauma may play a role. Injury to the kidney causes bleeding. It is more probable that latent stones begin to give trouble after trauma. It is only hypothetical whether there is such a thing as Meckel's 'stone-forming catarrh.' " Israel cites "a case due to stenosis following lateral fistula due to injury to the ureter by a clamp during vaginal hysterectomy. Another case had colic following misplacement of the ureter by a vaginal fixation. Therefore, one should always remember that a former gynecological operation may be the cause of kidney colic. The cramp-like pains due to the rare condition of primary ureteritis cannot be diagnosed from kidney stone colic. A diagnosis is practically impossible when the colic is due to the form of ureteritis in which small concretions are passed, these having been formed in the ureter and having for their nucleus some small organic mass. Colic due to primary stricture of the ureter is very rare."

Benjamin R. Schenck,[7] speaking on the etiology of ureteral calculus, states: "Calculi formed primarily in the ureter are very rare. While they may result from urinary deposits about the site of an old infection or ulceration they more frequently occur after operations on the ureteral walls, as when a non-absorbable suture becomes the nucleus of an irregular deposit of phosphates or urates.

"The presence of a foreign body, such as a calculus, beginning its descent must cause a spasm of the muscular coats, and this we consider an important factor in causing the arrest of the stone. Added to this we have, in cases where infection is present, inflammatory reaction and swelling of the mucosa, which later cause thickening and rigidity of the ureteral walls and finally stenosis at the point of arrest. Such, as far as is known, are the general etiological factors."

R. C. Bryan,[8] speaking of the etiology of ureteral stone, states: "The possibilities of transmural bacillary infection must be considered as a constant menace to the ureter integrity, particularly if its mucosa has been insulted by the frequent passage of a concentrated urine loaded with irritating crystals such as oxalate of lime, or if indeed its wall has either mechanically or by processes of inflammation been restricted or damaged, as may be instanced in childbirth, appendicular, ovarian, tubal and peritoneal exudates, and consequent fibrotic replacements."

He also says: "As another and more recently developed causation of primary ureteral stone must be mentioned *stricture*. The acquired variety has been briefly mentioned above. In regard to the *congenital*, Bottomley[9] records 56 cases of congenital stricture of the ureter. Such a stenosis, if complete, would result in atrophy of the kidney or hydronephrosis. If of small caliber there would be, proximally, a dilatation and thickening of the ureteral wall. The opportunity here, through stagnation, for stone building would seem evident and is directly comparable to stone formation in a bladder behind an obstructing prostate."

Guiteras[10] considers most ureteral stones to be secondary as the rare primary stones are of a phosphatic origin and small.

J. M. Thompson Walker[11] says: "Very rarely a calculus is formed in the ureter itself." He gives examples of calculi formed around sutures and other foreign bodies.

John H. Watson, Surgeon to the Victoria Hospital, Burnley, in his paper on "Ureteral Stone,"[12] states: "There are, however, certain general facts which may be well worth recalling in these cases. In the first place stone in the ureter is certainly more prevalent than is supposed—according to some writers it is of greater frequency than renal calculus. They may arise in the ureter, but, as a general rule, they form in the renal pelvis and then pass into the ureter; the etiology is therefore practically that of renal calculi, which is still a vexed problem."

Braasch and Moore,[13] under the section on Treatment, state: "In the consideration of ureteral stone its renal origin should be borne in mind." Further data from this valuable contribution will be referred to later.

O. S. Fowler,[14] of Denver, has called attention to the possible importance of stasis in the etiology of stone formation. He attributes the stasis to a kink in the ureter, due to prolapse of the kidney, as demonstrated by the method he has done so much to develop of taking pyelo-ureterograms in the erect posture. Most of his roentgenograms, however, reveal ureters which are dilated below the point of kinking as well as above, and I would interpret them as being cases of unrecognized ureteral stricture low in the channel, causing a dilatation of the ureter and kidney pelvis and a prolapse of the heavy hydronephrotic kidney, resulting in apparent kinks in the upper ureter. I have several ureteral stricture cases showing this kidney prolapse and apparent ureteral kinking, but they all show the ureteral dilatation down to the stricture area, as illustrated strikingly in Figs. 1 and 2. Reference to Fig. 3 shows a remarkable circular twist of the upper ureter at the ureteropelvic juncture. This patient had a kidney fixation six months before this pyelo-ureterogram, but continued to suffer with renal colic. Viewing this picture one might say that the renal attacks continued because of faulty fixation of the kidney, resulting in this pelveoureteral twist, but the patient's symptoms were entirely relieved after dilatation of the two strictures in the lower ureter.

Reference to Fig. 4 shows a decided displacement and kinking of the right ureter in the appendix region and stasis above, which might be interpreted as ureteral obstruction, due to an old appendicitis: but reference to the legend will show that this was probably a patient suffering with the condition which I have described as simple stricture of the ureter.[15]

While Rovsing[16] was one of the early modern writers on ureteral stone, I have reserved quotations from him until the

last because of the clear manner in which he states that ureteral stone may form in a stricture.

He affirms that a stone may arise in the kidney and become obstructed in a narrow portion of the ureter, or it may arise primarily in the ureter, "due to a stasis of the urine by a stricture, be it a congenital stricture or valve formation, be it the result of tuberculosis or other suppuration and ulceration processes."

He takes the position that an infection is present at the same time which results in the deposit of hard urinary particles about the inflammatory organic elements.

Of the 16 cases of ureteral stone which he had observed, he was ready to assert that but one had certainly had its origin in the ureter itself. This was in the case of a woman who had worn an unsanitary pessary which Rovsing considers to have caused pressure on the ureter, resulting in a narrowing and inflammation, above which the stagnated infected urine could deposit its phosphates around the pus cells and fibrin shreds.

Rovsing takes up the consideration of the factors which may hinder a concrement which forms in the kidney and gets into the ureter from passing entirely through the ureter. He mentions as a physiological factor the three normally narrow areas in the ureter, and the pathological factors he divided into two groups, the intra-ureteral and the extra-ureteral.

In the first pathological group belong congenital valve formations or narrowings of the ureter, and inflammatory stricture, be it of simple or of tuberculous origin. Rovsing finds the suggestion in the literature that the passage through the ureter of sharp-edged concrements may wound the ureter and thus set up stricture formation and constriction for the stoppage of later concrements. He doubted whether any of his cases belonged to this class.

The extra-ureteral pathological processes which may cause the arrest of a stone may be tumor formations which compress the ureter or disease of the surrounding tissues. The

ureter may be situated in the contracted connective tissues and be compressed by them or it may be retracted laterally to form a kink and consequent blockage of a stone. He considered 2 of his cases as belonging to this category. In 1, a woman, aged forty years, had a severe illness eighteen months before her first ureter colic. This he interpreted as being due to a right-sided tubal pregnancy, resulting in an exudate behind and to the right of the uterus, displacing the uterus toward the right and leading to a sharp kinking of the left ureter, in which he demonstrated a stone. In a second case he found pyuria and a finger-sized calculus above the cervical portion of the uterus in a woman who had had puerperal fever, with peritonitis attacks, and had been confined in bed for six months.

Statement of the Author's Views. Except for the one factor of the intermittent gush of urine, which tends to sweep any foreign material from the ureteral lumen, a ureteral stricture presents the ideal conditions for the formation of calculus. The strength of the normal gush must be increased in the narrowed stricture area, and yet there must be some interruption and eddying of the stream in some cases, thus making a degree of stasis favorable to stone formation. Our histological studies of the stricture area have shown a change in the type of epithelium from the stratified transitional to the squamous, and that in some strictures there is actual ulceration. The urinary analysis shows blood in a fairly large percentage of stricture cases. This blood in some cases is probably due to overdistention and trauma to the kidney pelvis when the stricture swells shut, and in other cases it is undoubtedly due to bleeding from the ulcer at the stricture area. (See Figs. 5, 6 and 7.) A slight oozing and coagulation to form a small blood-clot at the stricture site should form an ideal nidus for the deposit of urinary salts, particularly with the added factor of stasis of the urine.

Without actual ulceration of the stricture mucosa we probably have other factors favoring stone formation, such as an excess of a serous or mucoid secretion, as found elsewhere

in catarrhal conditions of the mucosa, an increase in the desquamation of epithelium, and irregularities of the surface to favor the retention of these organic exudates and the deposit of inorganic precipitates.

It is probable that these mechanical theories do not fully account for the formation of stone in the ureter.

If they were all sufficient we would find ureteral stone in a much higher proportion of stricture cases than we do, and, furthermore, we would expect to find stone in the bladder much more frequently. In many cases of bladder inflammation which come under observation we have the ideal mechanical conditions for stone formation, such as ulceration, bleeding, increased exudation of serum and of epithelial cells and a comparative stasis of the urine.

In a very small percentage of bladder ulcers do we find stone formation. It is apparently favored when the urine carries a proteus or some other alkaline-producing organism, but the bladder ulcer may be covered with urinary salts in the presence of an acid urine and colon bacillus infection. Whenever we find a bladder ulcer covered with fibrinous and mucoid material impregnated with urinary salts we have potential stone formation, and it is probably the constant activity of the bladder walls plus the free drainage through the relatively large urethral canal that determines the breaking off and extrusion of these potential calculi.

Similar factors of peristalsis and force of the urinary stream tend to clear out incipient ureteral calculi, but the narrowed channel at the site of the ureteral stricture tends to hold these calculi and determines a much larger number of stones in the ureter as compared with the bladder.

While the presence of an infection probably favors stone formation by intensifying inflammation, causing more serous and epithelial exudate, and by decomposing the urine and setting free its solid constituents, and at times by furnishing clumps of organisms as a nucleus for stone, we must admit that we find many stones in the urinary tract unassociated with infection. It might be argued, as held by Harris, that

infection was present and was a factor in the laying down of the stone nucleus and that the infection cleared up later. We believe the presence of a stone in the tract would tend to perpetuate an infection, and in many of our stone cases there has been no past history suggesting a pyelitis or a cystitis. It is true that a patient with stone may have had an unrecognized pyelitis or cystitis attack, the symptoms of which were not marked and have been forgotten by the patient when she presents herself with evidences of aseptic stone in the tract.

Our conclusions are therefore that ureteral stricture plays a role in stone formation of far greater importance than has been realized heretofore. The majority of ureteral stones probably form within a stricture area. Some of these escape and give rise to bladder stones or are passed. Some stones formed in a stricture area probably escape and float upward and become kidney stones.

While we have in ureteral stricture the ideal conditions for stone formation considered from the "mechanical" viewpoint, we must credit the presence of "bacterial infection," with a share in the formation of some calculi; and it is probable that the "chemical" or "specific diathesis" theory cannot be eliminated if we are to account fully for this pathological phenomenon. We see an occasional patient who repeatedly passes uric-acid calculi from the kidney in whose urine we are not able to detect unusual changes and in whose urinary tract we are unable to demonstrate any abnormality during life. Such cases might be credited to a low-grade nephritis, but we usually think of this type of stone formation as due to "faulty metabolism," obscure biochemical changes or some such poorly understood diathesis.

The formation of the rare fibrinous calculi, according to Gage and Beal,[17] is probably preceded by a hematuria.

Clinical Observations Supporting the View that Ureteral Stones Often Arise Primarily in a Stricture. The extremely small size of many ureteral stones found encased in dense ureteritis tissue is an argument against the

old view that the ureteritis is due to the irritation of the stone. Such small stones if formed in the kidney would pass entirely through a normal ureter.

The occurrence of bilateral stricture with the presence of a stone only on one side argues for the secondary character of the stone. If the stricture formation were dependent on the irritation of the stone we would expect to find bilateral stone. Five of my cases have shown this condition and 3 of them are illustrated by Figs. 8, 9, 13 and 16.

Multiple stricture in one ureter with a stone in but one stricture area is suggestive of the strictures being primary, although one might argue that the stone caused one of these strictures and then passed up or down and set up a second point of irritation and ureteritis. Figs. 10, 11, 12, 13, 16 and 18-*c* illustrate three out of several cases of this kind under my observation.

Case 71 of my ureteral-stricture cases was a patient, aged thirty-seven years, who had suffered for eight years with left renal colic, the attacks becoming so severe recently that they required several hypodermics of morphin for their control. The urine was smoky with blood and contained pus and a colon bacillus infection. On passing a catheter prepared with small wax rings at intervals of 5 cm. for locating the possible ureter stone, and with one large wax bulb to dilate the ureter, the catheter returned with scratch marks on all the rings within 20 cm. of the tip. The eye of the catheter contained a particle of stone and the large wax bulb had embedded in its surface about a dozen particles of stone. The patient was completely relieved of her severe colic attacks and subsequently had several dilatations of the stricture area until her symptoms were relieved. She had the contracted type of pelvis, holding but 4 c.c. Scratch marks were never obtained after the first treatment, which seemed to have entirely cleared out the incipient stone formation.

In ureteral stricture case No. 13, seen with Dr. Pancoast, there was an obstruction in the ureter 6 to 7 cm. above the bladder, and on two occasions definite scratch marks were

obtained on the wax tip and bulb. A tender nodule could be palpated in line with the ureter, and on vaginal ureterotomy, seven days after the second ureteral dilatation, a dense scar-tissue area was opened but no stone was found. Dr. Pancoast then explored and drained the large hydronephrotic kidney, and at subsequent dilatations of the patient's stricture area, scratch marks were not obtained. It is probable that the patient had either passed her calculus before our operation or the stone was so small that it escaped our notice while we were doing the vaginal ureterotomy, or the stone had floated up and after operation escaped through the nephrotomy wound.

The large ureteral stone, Fig. 18-*d*, was removed from the right broad ligament region in my ureteral stricture case, No. 9, five years after I had repeatedly dilated strictures in the right broad ligament and iliac-gland regions and in the left broad ligament region. Figs. 14 and 15 show the condition found in the kidney three years after removing this broad ligament stone, and it might be argued that this stone originally formed in the kidney and got caught in the lower ureter, where it took on the ureteral stone form.

A stone having formed in a stricture area may persist for years with comparatively few symptoms, due to the fact that the urine tends to channel one side of the stone and keep a free passage (see Figs. 16 and 18-*c*.) Such a channelled stone may shift its position, due to a fall or some form of sudden exertion by the patient, when the lumen may be blocked and a typical sudden colic supervene. Such a sudden obstruction, if unrelieved, leads to serious consequences to the already partially damaged kidney, and it may lead to reflex anuria and death of the patient, particularly if the opposite kidney be below par from the effects of stricture or stone in its ureter or from any other cause.

The sudden shifting of position and consequent blockage may result in such a head of pressure that the stone is swept out of the ureter, and becomes a bladder stone, or is passed to the outside. Figs. 19 and 5 illustrate cases in which the patients had passed ureteral stones but continued to have

symptoms until dilatation of their ureteral strictures. In my ureteral stricture case No. 23 the patient had suffered with bladder discomfort and incontinence for nine months, and I removed a stone 15 mm. in diameter from the bladder. Six or eight years previously the patient had suffered a severe colic attack on the left side, requiring morphin. The *x*-rays showed in addition to the bladder stone a small shadow in the left broad ligament region, and investigation with the wax-tip and wax-bulb catheter demonstrated this to be a phlebolith, but the patient had two strictures in the broad ligament region and a left colon bacillus pyelitis, with kidney content of 21 c.c.

It is likely that the records of a stone taking many years to pass or to reach a certain point in the ureter before its removal by operation are often records of patients who have suffered for years with ureteral stricture and have more recently developed a stone in the stricture area.

The comparatively frequent association of kidney stone with ureteral stricture (5 cases in my first 50 ureteral stricture cases, see Figs. 20 and 21) convinces me that some stones forming in a stricture may eventually become released, float upward and become kidney stones. Such stones may remain in the kidney permanently or they may be migratory, being found either in the kidney or ureter; or they may increase in size while in the kidney pelvis and eventually begin a descent and become blocked in some portion of the ureter above the stricture site, or make their way back to the stricture site, where they completely block the ureter because of their increased size and lack of lateral urinary channel, such as we usually find in those stones which have always remained in the stricture area.

I believe that this peripatetic character of some stones explains some of the problems suggested by Braasch and Moore, whose report on the vast experience and accurate observations of the Mayo Clinic furnish us with one of the most valuable chapters on ureteral stone. In their paper above referred to they state: "The majority of the stones in

the pelvic portion were not lodged exactly at the point of narrowing at the ureterovesical juncture but a short distance above it." My work on ureteral stricture has shown that the stricture occurs in the broad ligament region or within 6 cm. of the bladder in 92 per cent. of the cases. By far the greatest proportion of these strictures were at a point 3 to 4 cm. above the bladder rather than in the ureterovesical juncture.

Braasch and Moore state that of those stones described at the ureteropelvic juncture the majority "were found a short distance above the point of narrowing." Contrary to the past literature on ureteral stricture, which has considered this lesion of congenital origin and located it at the points of congenital narrowing in the ureter, I have shown that stricture in the pelvic brim region does not occur at the ureteropelvic juncture but at the iliac gland region 3 or 4 cm. below the pelvic brim (17 per cent.). Some of these stones, which Braasch finds a short distance above the pelvic brim, were probably formed in the kidney, and some were probably formed in a ureteral stricture and had travelled back to the kidney, where they increased in size and on returning to the ureter they were too large to reach the pelvic brim. The same is probably true of the group of which he states: "It is difficult to explain the large number of stones found in the upper third of the ureter." He states: "The majority of such stones, however, were of considerable size." He also states: "The average size found in the lower third of the ureter was much smaller than those found in the upper ureter" (these lower stones have always remained in the stricture areas). Braasch states: "As a general rule, stones situated in the ureter at points other than those of natural narrowing were larger and caused more renal destruction than the others" (stones which lack the lateral urinary channel so often found in those stones permanently fixed in a stricture area).

Practical Clinical Deductions. This study was undertaken to emphasize the importance of ureteral stricture as an etiological factor in the formation of urinary calculus.

A few clinical deductions following from this thesis are of such practical importance that it seems appropriate to give them space in this paper devoted to etiology. Ureteral stricture is of such frequent occurrence that every case demonstrated by *x*-rays to have a small stone in the kidney pelvis, should have investigation for the presence of ureteral stricture before operation. (See Fig. 20.) In lieu of such preoperative investigation, such cases at operation should have a renal catheter, carrying a 4 mm. wax bulb within 3 cm. of its tip, passed through the pyelotomy opening downward into the bladder. Such a wax bulb will serve to detect and dilate an occasional stricture low in the ureter. If a stricture is discovered in this manner, drainage should certainly be left down to the pyelotomy wound to take care of possible leakage, due to the swelling shut of the stricture area incident to the trauma of its first dilatation.

The returning wax bulb should be investigated for scratch marks, for in rare instances an unsuspected ureteral stone will be indicated by this test.

The above instructions apply with equal logic to operations for ureteral stone located above the pelvic brim, particularly if the stone is found in a dilated ureter and is not encased in an area of dense infiltration (probably a migrating stone). In operating for a stone in the lower half or pelvic portion of the ureter one will usually find such stone in a stricture area located either a few centimeters below the pelvic brim (iliac gland region) or a few centimeters above the bladder (broad ligament gland region). In view of the comparative frequency of multiple strictures in the pelvic ureter, careful investigation should be made for other strictures than the one encasing the stone (Figs. 10, 11, 12 and 13). With a well-channelled stone it is more than probable that the symptoms are due to the second stricture, although one occasionally sees a case with marked symptoms, due to a single stricture with a well-channelled stone, the symptoms probably being due to a shifting of position by the stone.

On finding a stricture area about a stone this should always

be thoroughly dilated as a part of the operation. It is preferable, when possible, to make the ureterotomy opening in the dilated portion of the ureter above the stone and to then grasp the stone with delicate forceps and drag it up through this opening or to "milk" the stone out of its stricture bed and up to the ureter incision. Unless the stone is large enough to be well outlined in its stricture bed one is likely to have trouble in dislodging it, and in such cases it is better to cut directly into the stricture area and on the stone. The dilatation of the stricture must then be carried out in both directions from the ureterotomy opening.

In operating for a fairly large stone in a stricture area one occasionally finds a second smaller stone or a nest of small stones just above or below the large stone. Great care should be taken to prevent these smaller stones from escaping up or down in the ureter to cause future trouble.

With added experience we should get most small stones in the pelvic ureter to pass spontaneously. Fig. 17 shows the largest stone I have dislodged by this method. Fig. 23 shows the type of gum-elastic graduate dilating bougie I am now using, with much satisfaction, in the later treatments for ureteral stricture, and for dilating in the stone cases, after using my spiral tip wax-bulb catheter (Fig. 22), for the early work in testing for scratch marks, dilating the stricture, getting its location, the size of the kidney pelvis, lavage, culture taking and estimating the renal function. In dilating for stone one should avoid treating too frequently. My experience with ureteral stricture dilatation has shown that it takes from a week to ten days for the edema, incident to the trauma of treatment, to fully subside. This corresponds with my experience in getting ureteral stones to pass. They usually escape in from eight to ten days after the last dilatation. Occasionally, after the ureter has been previously stretched by repeated and increased caliber dilatations, a stone will come down within the first few days of the last treatment.

CASES SHOWN IN PLATES

Case I.—Mrs. B., aged twenty-eight years, referred by Dr. Bay, May 15, 1917. Attacks of pain in the right side for four or five years. First attack came suddenly and lasted two or three hours. Two or three attacks within a month, then none for about a year, then awakened one morning at one o'clock with such a severe attack she could hardly get out of bed. For the past three years she has had two or three attacks a week but has gone for two or three months without an attack. There has been some pain in the side almost every day since the first attack. In November, 1916, Dr. Bay did an appendisectomy and fixation of right kidney. Four days after leaving hospital the patient had one of her typical attacks and continued to have them for six months until the strictures were dilated. The kidney holds 15 c.c. Two strictures, one at about the bifurcation of the internal iliac vessels and one within 2.5 centimeters of the bladder. Symptoms relieved after two dilatations. This patient's family physician, Dr. Henderson Irwin, of Eureka, North Carolina, after the experience with this one stricture patient, diagnosed a second case in November, 1917, and brought her to me for confirmation of his diagnosis and for treatment. He then reported this first patient as enjoying good health and having gained 16 pounds in weight in the seven months. (Fig. 3.)

Case II.—Mrs. W., aged thirty years, referred by Dr. Carroll, Wallace, North Carolina, July, 1917. Pain in the right back for the past fifteen months. Pain of a dull character most of the time with intermittent colics. For the past month almost daily severe colics, some of them requiring hypodermics of morphin. Pain in the lower right pelvis, reflected to the right hip and right buttock with frequency of voiding, the pain being worse rather than better after voiding. Never noticed blood in the urine. Note *x*-ray catheter obstructed a few centimeters above the bladder. Thorium picture shows some dilatation of the ureter from this point to the pelvic brim region where the ureter is drawn laterally into rather sharp angles and shows rather greater dilatation above this point. The kidney pelvis holds 15 c.c. One would be tempted to interpret this as a case of ureteral kinks due probably to an old appendix inflammation, but on later investigations we found four definite stricture areas, the highest one being 10 centimeters above the bladder or slightly below the pelvic brim and the patient's symptoms were relieved by dilatation of these strictures. In November, 1917, the patient returned with increasing symptoms on the left side and we found two stricture areas at points 7 and 4 centimeters above the bladder in the left ureter. This we consider fairly good evidence that the patient's condition was one of so-called simple stricture of the ureter and the apparent kinks at the pelvic brim region were not concerned with the original symptoms. (Fig. 4.)

Case III.—Mrs. L., aged twenty-nine years, referred by Jeffries Buck, in August, 1917. The patient had had pain in the left back for about one year. In April, 1917, about two days before her menstrual period, the patient had an extra amount of pain in the kidney region and then a terrible attack, lasting two hours, of pain throughout the left side and hemorrhage in the urine. This two-hour attack ended by the passage of a calculus. The patient felt the cutting of the stone in the urethra and heard it strike the toilet basin but did not recover the stone. Since April she has felt much better except for the forty-eight hours preceding each menstrual period, when there has been an increase of pain in the back and low in the left pelvis and a passage of much blood in the urine. Since the passage of the stone there has been a sensation of fulness in the bladder and diurnal frequency.

Examination revealed thickening of the broad ligament portion of the left ureter, tenderness and a desire to void on palpating this induration. Urine from the bladder, macroscopically clear; microscopically,

many erythrocytes, no leukocytes, no bacteria, no albumin. Wax bulb test demonstrated stricture 2 centimeters above the bladder. Injection test showed a kidney content of 6 c.c., a small pelvis. This thorium x-ray shows a small pelvis but a slightly dilated ureter. Three treatments with increasing sizes of wax bulb up to 5 mm. followed by complete relief. (Fig. 5.)

Case IV.—Patient, aged sixty-nine years, referred by Hardee Johnston, Birmingham, Alabama, admitted to the Church Home and Infirmary May 18, 1917. Since April 5, 1917, she has had four attacks of hematuria, each attack lasting four days. There is no pain but a slight uneasy feeling in the right flank during the attacks. A renal catheter enters the right ureter only 3 or 4 centimeters. Thorium injection beyond catheter of 13 c.c. Later two successful dilatations of stricture 3 centimeters from the bladder. Report October 1, 1917, no further bleeding and no symptoms. (Fig. 6.)

Case V.—Miss S., aged twenty-four years, seen in consultation October, 1917, with Dr. Casler, Scarlet fever at nine, followed by "catarrh of intestines," constipation and mucous stools. Gastric lavage weekly for three years by Dr. Friedenwald. At sixteen years of age, onset of attacks of pain in the right side, exaggerated at the menstrual period and accompanied by fever. The attacks occurred about every two weeks for a period of eight months, when Dr. Finney removed the appendix. The attacks continued but apparently in milder form and she thought they were due to the menstrual periods. In January, 1915, adenoids were removed, the tonsils having been removed the winter before her "appendicitis" operation. January 1, 1917, an attack of grippe followed by an exaggeration of her pain in the right side and constipated bowels. Dr. Fisher operated for intestinal adhesions January 27, 1917. In May, 1917, Dr. Casler discovered ureteral stricture with infected hydronephrosis. The patient had been afflicted with repeated crops of boils ever since her "appendix trouble" began. This skin trouble improved after Dr. Casler got rid of the patient's Staphylococcus aureus kidney infection by dilatation of the stricture and lavage. When I saw the patient with Dr. Casler she was still having pain in the right side at times and rather frequent hemorrhages with the passage of clots in the urine. With more extensive dilatations of the stricture area the bleeding ceased but the patient still has occasional attacks of pain. (Fig. 7.)

Case VI.—Miss G., aged twenty-six years, referred by Fletcher Jordan, Greenville, South Carolina. Tonsillitis since childhood. Intermittent renal colics on left side for one year, on right side for three months. Stone 3 centimeters from bladder on left side in dense stricture area. Dense stricture 3 centimeters from bladder in right ureter. Bilateral hydronephrosis. Urine not infected. Left ureter exposed by extraperitoneal inguinal incision, stone removed and stricture dilated. During convalescence the right ureteral stricture dilated by the vesical route and diseased tonsils removed. (Fig. 8.)

Case VII.—Mrs. C., aged forty-eight years. Severe renal attacks on right side for eighteen months, requiring much morphin. No symptoms on left side. Urine catheterized from bladder shows leukocytes, erythrocytes, colon bacilli and a trace of albumin. Stricture of right ureter 2 or 3 cm. from bladder. On catheterizing to obtain Fig. 9, we found unsuspected obstruction and dilatation of left side. The right side held 38 c.c. and the left 30 c.c. After withdrawing the catheter from the left side and finding scratch marks on the wax bulb and tip, the small stone shadow was discovered in the lower end of the left ureter at a point symmetrical with the stricture of the right side. Examination with wax tip bougie twenty-four days later showed that the stone had been passed.

Case VIII.—Mrs. R., aged forty-nine years, referred by Erasmus Kloman. For six years she had had intermittent colic-like pains in the

left side and back and radiating down course of ureter, accompanied by nausea and vomiting. After this plain x-ray demonstrating the shadow of a stone apparently located in the upper pelvic portion of the left ureter, Fig. 11 was taken. A No. 7 whistle tip catheter was prepared with wax rings and a large wax bulb and it was successfully passed to the kidney. About 2 ounces of urine escaped rapidly and 75 c.c. of thorium were introduced to the kidney for this x-ray photograph. On withdrawal of the catheter the wax bulb hung definitely in two areas, and the wax bulb and the small wax rings within 15 centimeters of the end of the catheter were deeply scratched on one side. The patient continued in her uremic and weakened condition with much nausea and vomiting, but had less pain after the passage of the wax bulb until the eighth and ninth days, when there was a great increase in pain. We hoped the stone was passing but the patient's condition was so threatening that we decided to operate (see Fig. 12), On the way to the operating-room Fig. 12 was taken, showing that the stone had left its original site and had caught in the broad ligament region, probably in a stricture area, as indicated by the second "hang" of the wax bulb. McBurney muscle-splitting extraperitoneal incision low over the left pelvis, the dilated ureter easily identified at the pelvic brim region and a tape passed under it. At the region of the internal iliac bifurcation there was seen a definite stricture area but the stone had escaped. The stone was found in the broad ligament region and was easily pushed back to the upper stricture area but could not be brought through this area into the more dilated portion of the ureter above the stricture until we made a ureterotomy opening above the stricture and dilated with Hegar dilators from sizes 5 to 8. Each dilator hung definitely at the stricture area. After this dilatation the stone was easily pressed up through the stricture and out of the 1 centimeter incision. A No. 6 Hegar dilator was passed all the way to the bladder to dilate the stricture area in the broad ligament region. The operation was done under laughing-gas and was finished in twenty minutes, causing only moderate shock. The patient had an output of 400 c.c. of bloody urine during the first twenty-four hours after operation, but she failed to rally and had much nausea. During the second twenty-four hours she secreted only 300 c.c. and died at noon of that day, being perfectly conscious to the last. This was probably a case of bilateral ureteral stricture in which the uninvestigated kidney was too damaged to carry the patient through after relieving the obstruction on the stone side.

Case IX.—Mrs. McD., aged thirty-three years, referred by H.A.Fowler, of Washington, in June, 1917. First childbirth ten years ago, a forceps delivery followed by bilateral phlebitis. In bed with this two months and then developed an attack of pain through the left hip region which her husband (a physician) thought was due to the passing of a renal calculus. The pain ceased after a hypodermic of morphin and it was supposed the calculus had passed. The urine was clear at this time. Within the next year or two she had several similar attacks and then they ceased for six or seven years. About two years ago she was in bed for three weeks with an attack of illness with fever, but no pain. The urine then showed blood and pus. She went to Philadelphia after this attack and Dr. Pancoast found two ureteral stones by x-ray. Dr. Keene verified this, saying the lower stone was within two inches of the bladder. Dr. Fowler verified these findings and wrote "that in the first plate we made the two shadows were touching each other. In some later plates the upper shadow had moved upward and was separated considerably from the lower shadow. It would seem that the large upper stone moves up and down in the ureter and is not fixed as the lower one appears to be."

My examination revealed tenderness in both broad ligament regions on palpating the ureters. What seemed to be a small stone was easily palpated about 3 centimeters from the bladder on the left side. Inves-

tigation of the right side showed a definite stricture 3 centimeters above the bladder and the reaction from this examination kept the patient ill for a week. Investigation of the left side resulted in obstruction to the *x*-ray catheter 3 or 4 centimeters above the bladder. An attempt to inject the kidney and upper ureter with thorium resulted in much pain in the lower ureter after 1 or 2 c.c. entered the ureter. Note the upper shadow (thorium) and the small amount of thorium which has run back into the bladder. (Fig. 13.) Operation revealed but the one small stone wedging the end of the catheter. This was encased in a broad ligament stricture and there was a second dense stricture at the iliac gland region, which had trapped the thorium, caused the intense ureter pain, and resulted in the upper large shadow taken to be a second stone. The stone was removed (see Fig. 16), both strictures were well dilated, the upper ureter was followed to the kidney pelvis with negative results; *x*-ray was taken during convalescence with negative results. The patient wrote three months later: "I have never been as well in my life, and have gained 25 pounds."

Case X.—This patient first seen March 24, 1909, when she was almost exsanguinated from hemorrhage from the right kidney. Nephrotomy March 26, draining an acute Staphylococcus aureus pyelonephritis kidney, and control of bleeding by a square catgut suture tied about a gushing point of hemorrhage situated in the pelvis immediately beneath a papilla in the upper pole. The patient had always suffered with tonsillitis, having had an acute attack three weeks before the acute hemorrhage.

Six months later, discovery of two strictures in the right ureter, one in the broad ligament region and one in the iliac gland region. Right hydronephrosis of 15 c.c. One year after the operation, discovery of two strictures in the left ureter and a left hydronephrosis of 30 c.c. with Staphylococcus aureus infection. After dilatation of the strictures the patient gained in weight from 117 pounds before the operation to 164 pounds and appeared to be in excellent health, although the infection persisted. In December, 1914, I removed the large ureteral stone (Fig. 18, *d*) and made a wide dilatation of the right ureteral strictures. *X*-rays by Dr. Baetjer on November 26, 1910, were negative for stone on either side. On December 29, 1917, this patient again had signs of obstruction on the right side. Fig. 14 was taken. Catheterization with wax-tipped bougie gave scratch marks, and the stone at the pelveo-ureteral junction was evidently loosened, as the patient became comfortable after the examination and her temperature of 103° dropped to normal. Phenolsulphonephthalein injected into the vein yielded color reaction from the left side, collected through the bladder, in eight minutes and from the right side through the catheter in twenty minutes. In one-half hour the left side secreted 27 per cent. and the right side 3 per cent. Later investigation of the left side with a 4 mm. wax bulb catherer showed the strictures well open, the kidney pelvis to have shrunken from 30 to 10 c.c. and a staphylococcus infection still present, but without leukocytes (see Fig. 15). The patient developed a severe attack on January 11, 1918, and right nephrectomy was done January 12. The large stone seen blocking the upper ureter in Fig. 14 had slipped down and become wedged a short distance above the pelvic brim. The ureter was tied and cut below this stone. Most of the stones in the kidney were extruded while removing the necrotic adherent kidney and they were arranged for this photographing (Fig. 15) as nearly as possible in their original positions seen in Fig. 14.

Case XI.—Patient, aged forty-seven years, had passed a navy-bean-sized ureteral stone five years previously. Complained chiefly of pain in the left flank and pressure in the rectum. Failure of catheter to go beyond the pelvic brim region at first examination. Successful catheterization at all later attempts and dilatation of a stricture, just below the pelvic brim with the wax-bulb. Rapid shrinkage of pelvis of the kidney from 28 to 10 c.c. and relief of symptoms. Observation five months later; pelvis of kidney held 10 c.c. (Fig. 19).

Fig. 1.—To illustrate picture often seen in roentgenography and heretofore interpreted as hydronephrosis due to ureteral "kinks." X-ray catheter obstructed in a kink. Note the dilatation below the kinks, showing that the real cause of obstruction is lower. The symptoms in these cases clear up without fixation of the kidney after thorough dilatation of the strictures and establishment of free urinary drainage. Fixation of the kidney without dilating the stricture is followed by further symptoms.

Fig. 2.—Same patient as illustrated in Fig. 1, the catheter being withdrawn until the wax bulb, located 10 centimeters from the tip of the catheter, has obstructed in the upper stricture at the iliac gland region. Note slight dilatation of the ureter between this stricture and the lower stricture in the broad ligament region.

Fig. 3.—Apparent twist in ureter at kidney pelvis. For description of this and following plates see cases reported at end of article.

Fig. 4.—Ureteral stricture; displacement of ureter in appendix region.

Fig. 5.—Illustrating case with periodic hemorrhage probably from a stricture area.

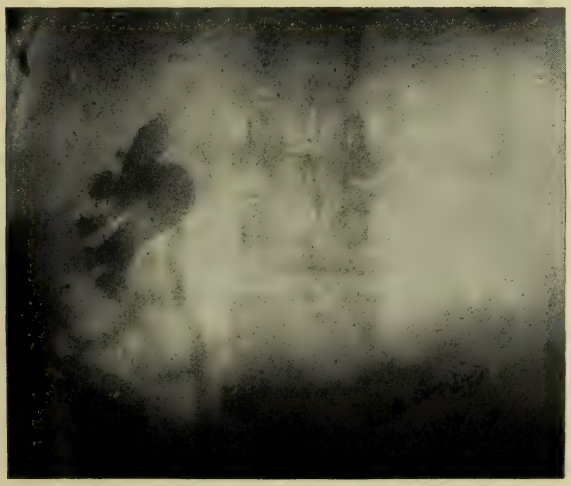

Fig. 6.—Ureteral stricture with hematuria.

Fig. 7.—Ureteral stricture with hematuria.

Fig. 8.—Bilateral stricture and stone in one stricture.

Fig. 9.—Bilateral stricture with stone in one stricture.

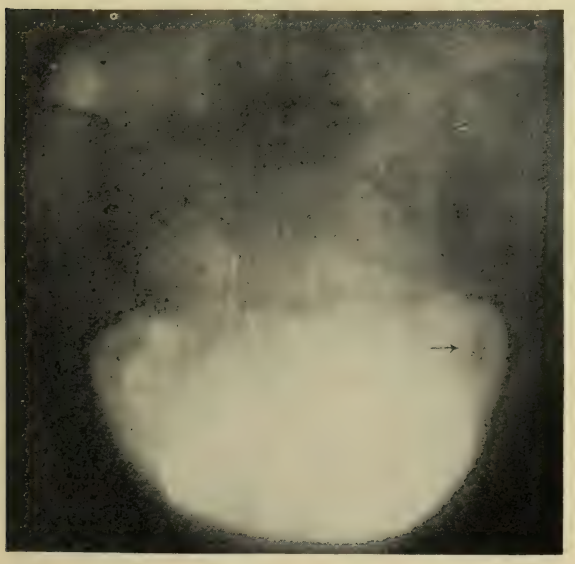

Fig. 10.—Multiple strictures and stone in upper stricture.

Fig. 11.—Same patient as Fig. 10.

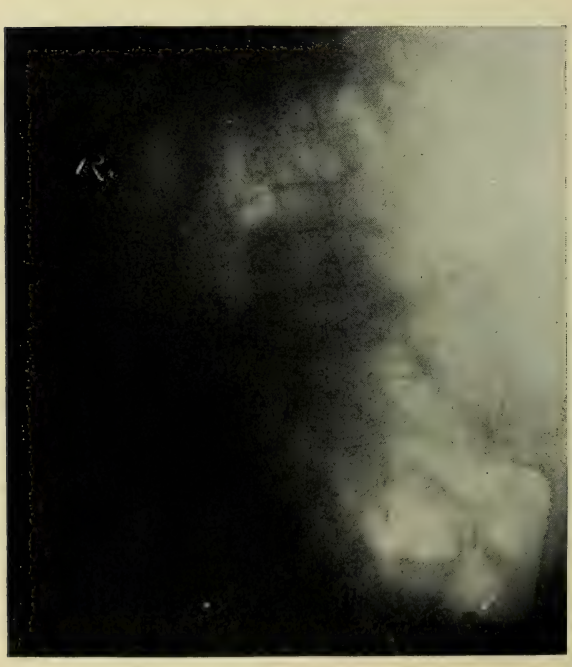

Fig. 12.—Same patient as Figs. 10 and 11.

Fig. 13.—Illustrating a case with two strictures in the pelvic portion of the left ureter; a stone in the lower stricture obstructing the catheter; shadow of thorium dilating ureter between the two strictures. Recently developing symptoms from right ureteral stricture.

Fig. 14.—Recent *x*-ray of a ureteral stricture case.

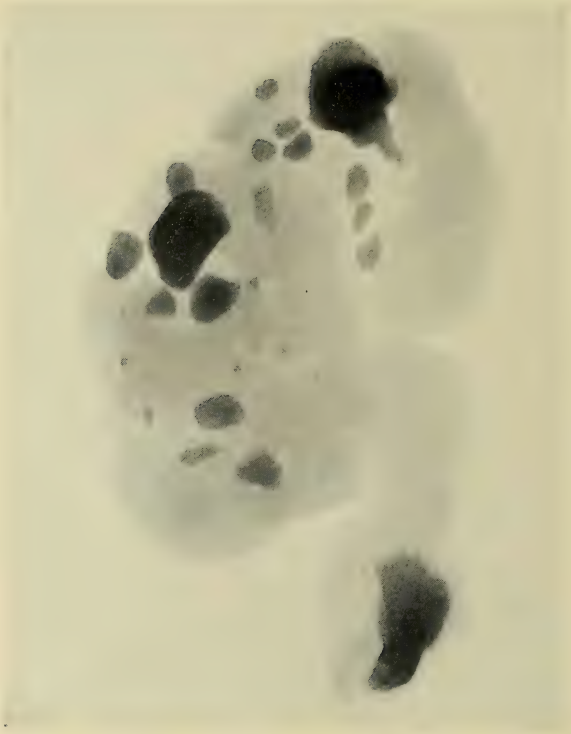

Fig. 15.—Removed kidney and upper ureter from patient illustrated by Fig. 14.

Fig. 16

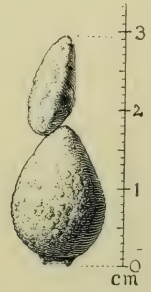

Fig. 17

Fig. 16.—Stone from Case IX, illustrated in Fig. 13. Note depth of lateral channel for free passage of urine. It is probable that the patient's symptoms came from the obstruction by the upper stricture rather than from this stone situated in the lower stricture.

Fig. 17.—Stones passed spontaneously ten days after dilating the ureter with a No. 16 ($5\frac{1}{3}$ mm.) gum elastic bougie. Increasing sizes of bougies had been used in 7 treatments at intervals of two weeks. These stones were passed by a stout patient who had been operated upon one year previously for the removal of abdominal fat, and it was particularly gratifying to avoid a second operation in her case.

FIG. 18.—*a*, gall-stone; *b*, appendix stone from one of the author's ureteral stricture cases, whose symptoms were due to ureteral stricture; *c*, ureteral stone from another stricture case, showing deep channel for free passage of urine, the patient's hydronephrosis symptoms probably due to a dense ureteral stricture located 3 centimeters below the stone and about 3 centimeters above the bladder; *d*, ureteral stone removed from stricture area five years after treatment of ureteral stricture (see Figs. 14 and 15).

FIG. 19.—Ureteral stricture, case.

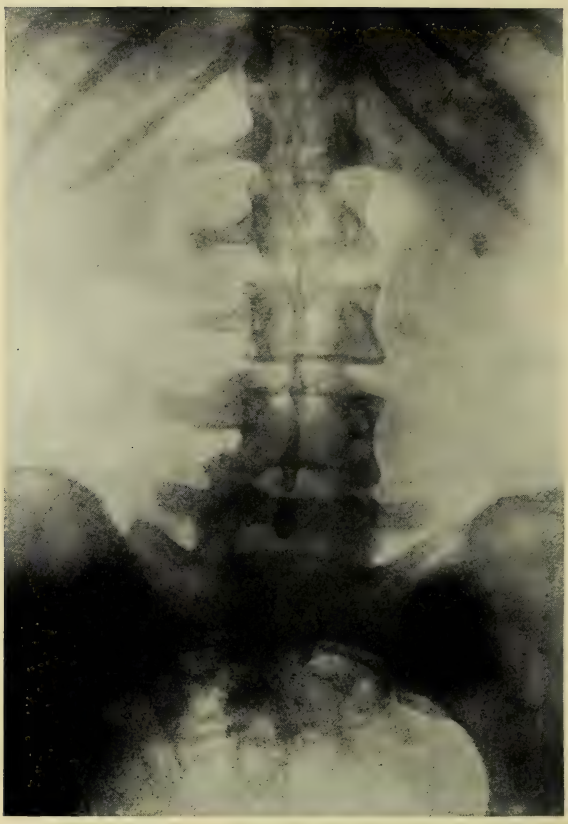

Fig. 20.—Ureteral stricture, suffering with left kidney colic several years. The *x*-ray revealed a stone in the pelvis of the kidney. Investigation with the wax-tipped catheter accidentally led to the discovery of ureteral stricture 7 centimeters from the bladder, the kidney pelvis holding 8 cubic centimeters. Complete relief of symptoms after dilatation of the stricture, and the patient refused operation in spite of being shown the *x*-ray of her kidney stone. This case probably explains the occasional case seen by the surgeon in which pyelotomy for stone is followed by urine drainage for a disappointing length of time, and after healing the patient continues to have symptoms similar to those before complained of.

Fig. 21.—Showing obstructed x-ray catheter in right ureter and stone in right kidney pelvis. Ureteral stricture case 50. Age thirty-two years, the only case in about 300 with stricture near the kidney. Catheter diverted laterally just above the pelvic brim and stopped at this same point in roentgenograms taken on three different days. Could not inject sufficient thorium into the kidney to get a definite shadow of pelvis. Nephrectomy; stricture immediately below enlarged pelvis would not take the fine silver probe. The patient had suffered attacks since early childhood, the pain beginning in the appendix region; and as it grew more severe it traveled diagonally across the abdomen and settled in the left kidney region. Operation three years previously, removing a diseased appendix and chronic pelvic inflammatory trouble, but without influencing her chief symptoms.

Fig. 22.—Graduate gum elastic dilating bougie, 38 centimeters long, the pointed tip gradually increasing in size until full diameter is reached about 15 centimeters back of the end. Sizes 18, 20 and 22, $7\frac{1}{3}$ millimeters diameter, are most useful. This dilator is useful for the final dilatations of ureteral stricture and for dilating to bring down stones in the pelvic ureter. The early investigations for stricture and stone are made with the spiral tip, wax-bulb renal catheter, Fig. 23.

Fig. 23—Renal catheter with "cork-screw" or spiral wax-tip and spindle-shaped wax-bulb. The pure beeswax is used. For the first investigation the bulb should be small, 3 or 4 millimeters in diameter, and may be increased gradually in size to 6 millimeters.

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RECURRENCE OF STONE IN THE KIDNEY

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The recurrence of a condition for which a surgical operation has been performed is an important matter and merits serious consideration. The publication in 1915 by Cabot and Crabtree[1] of a study of end-results in operations for kidney and ureteral stone, performed at the Massachusetts General Hospital for a period of eight years previous to January 1, 1914, was somewhat startling. In this it was shown that 49 per cent. of the cases suffering from kidney stone and 29 per cent. of those suffering from ureteral stone recurred. In 1917 Braasch[2] and W. J. Mayo[3] published a review of 450 patients operated on at the Mayo Clinic for nephrolithiasis, and from a careful analysis of these cases the recurrences were found to be slightly less than 10 per cent. The question of recurrence of stone in the kidney was brought somewhat forcibly to my own attention by having 4 cases under treatment at the same time. These happen to be the only cases in my practice that have come back, but there were doubtless others from which I have not heard. In view of the two very large series referred to above, my individual statistics would hardly add much to the subject. However, as each of my cases shows a different type of recurrence, following a different operation, the histories themselves should be of interest.

In dealing with stone in the kidney I have followed Brewer[4] in dividing my cases into two groups: one in which the stone was apparently the primary condition and the second where

the infection of the kidney was apparently the primary and predominating element. In the first case the removal of the stone was the indication, in the second the cure of the infection, which would usually mean the removal of the kidney, as the infection is ordinarily too far advanced and the alteration and destruction of the kidney too permanent to hold out much hope of improvement from any form of conservative treatment. Whether this simple classification meets the conditions, further investigation must prove.

There can be no question about the soundness of the three principles of treatment laid down by Schede[5] when he states that the object of treatment is, in the first place, to remove a formed calculus; in the second place to limit the injury thereby produced so far as possible; in the third place to protect the patient against a return of the trouble. The first two have been amply met by the refinements of modern technic, but the third has apparently received very little attention. Previous to the publication of Cabot and Crabtree's paper there seems to have been no systematic effort to determine whether the trouble returned or not.

It is not the purpose of this paper to enter into an exhaustive discussion of any part of the subject but simply to use the cases reported as a text for comment.

1. *The Cause of Formation of Stone.* In order to prevent a recurrence it would be necessary to understand why stones form in the first place. Prof. Schede[5] (1904) describes the formation of calculus as it has been usually understood, commencing with a nucleus of some sort and the deposit being made in concentric layers. He speaks of an organic framework, demonstrated by Ebstein, of the investigations of Moritz, which showed that this organic framework was present in all urinary crystals, but concluded that the quantity of this organic matter may be a factor. He also thinks that the composition of the urine, retention, geographical situation, customs and habits and period of life are important. On the other hand Kelly and Burnam[6]

(1914) state: "The essential conditions which lead to stone formation are imperfectly understood. In regard to age, sex, habits and diet none of these seem to play a great part. The stones which are found in the kidney are composed of substances normally present in the urine. The problem to determine is why these salts are precipitated into stones in some cases and not in others." In reference to nucleus they state: "It has been suggested again and again that foreign elements in the urine, such as bacteria, blood clots or shreds of tissue, may furnish the nuclei on which stones are built." Numerous cases are on record in which bacteria, blood-clots, etc., have been found in the nuclei of stones. Nevertheless, such findings are the exception. Barker[7] (1916) defines a renal calculus as "the formation in the pelvis of the kidney or in the kidney itself of calculi through the deposition of solid substances from the urine, usually deposited upon some organic nucleus;" and in reference to etiology, "many studies have been undertaken to determine the cause of stone formation in the pelvis of the kidney, but the exact pathogenesis is still *far from clear.*"

2. *The Effect of any General Treatment Directed to the Prevention of Stone Formation.* The treatment advised for lithiasis is well illustrated by the article on this subject in Forchheimer's *Therapeusis* by F. S. Meara[8] (1913), who gives many directions concerning exercise, alcohol, water and diet, and in reference to the latter describes various diets depending on the composition of the stone. This is somewhat confused by the fact that the majority of stones are of mixed formation, and he is then forced to quote Klemperer, who says: "The prophylaxis of renal calculus lies in the mixed diet dictated by nature." I have had no experience in dieting to prevent stone or its recurrence, but in one of the cases which I report the patient was directed to drink large quantities of water, which he did religiously, and notwithstanding this he had two recurrences. It would thus appear that we have at the present time no definite method of combating stone formation.

3. *The Role Played by Infection.* The case of Mrs. R. C. A., which I report, would appear to be one of a long-standing infection with secondary stone formation. Infection, I believe, will have to be considered as very important in considering stone formation and its recurrence. In the 30 cases of stone which were chemically investigated by Dr. G. L. Gordon and reported by Kelly and Burnam,[6] it is stated: "It is equally apparent that in mixed stones the phosphates ultimately predominate." The deposition of phosphates is certainly in the vast majority of cases associated with infection. This is a matter of common observation, and is due to the resultant change in the reaction in the urine. It is now generally accepted that infection of the kidney is hematogenous in origin, the kidney being one of the organs through which bacteria are excreted. It would appear that the alterations in the kidney as the result of infection may be responsible primarily for certain stone formations, and in other cases the presence of a stone may be the predisposing cause of an infection, which will be the cause of much of the symptomatology, and certainly of the destructive action on the kidneys. Mayo states that the type in which recurrence is most apt to occur is in "persons having large and branched stones located in a hopelessly damaged and infected kidney in which a conservative operation has been performed;" and in cases in which it is necessary to conserve the kidney he lays great stress on the method of drainage. This of course is done to overcome infection and prevent pocketing. In one of the cases which I report an attempt was made to conserve the kidney because the functional test showed that the affected kidney had a good function notwithstanding the presence of stone and infection. This was confirmed by incision and inspection of the kidney cortex at the time of operation. There was no recurrence in the kidney operated on but a prompt recurrence in the kidney of the opposite side.

4. *The Anatomical Cause.* Frequent mention is made of the fact that when stones recur they recur in the same loca-

tion in which they were first found. In one of the cases reported by me there were two recurrences in identically the same location following two operations on the same patient.

5. *Effect of the Type of Operation.* No improvement can be made on the rules laid down by W. J. Mayo.[3] Notwithstanding this, however, one of the cases reported by me showed a prompt recurrence in the opposite kidney after a nephrectomy, in the same kidney after a pyelotomy and none in the kidney on which a nephrotomy was done, but prompt recurrence in the opposite kidney.

6. *Recurrence is Evidently More Frequent than Supposed.* This is due to the fact that until recently methods of determining recurrences have not been exact, and also to the optimism of surgeons who after they have operated on a case are convinced that the patient is entirely well and pay no attention to their complaints. The time has about come now, however, when a good many of these cases that have been operated on for kidney stone are beginning to turn up. It is to be hoped that these cases will be carefully studied to determine why they complain and if the stone has recurred. Cabot and Crabtree have established as the standard of a cure a negative physical examination, so far as the urinary tract is concerned, a urine that is normal on chemical and microscopic examination and a negative *x*-ray. It would appear to me, as has been suggested by Braasch, that the presence of blood and pus in the urine may be the evidence of a pyelitis which had never been cured and which may have been the causative factor of the preëxisting stone. It would hardly be fair to count this as an evidence of recurrence.

7. *Patients may have Stone and Still Be in Comparative Good Health even when there has been a Recurrence.* This is evidenced by two of the cases reported by me. In one in which a nephrectomy had been done and a stone had recurred in the opposite side the patient had a large quantity of pus, diminished kidney function and some edema and general bad health. She had been treated with various urinary

antiseptics and irrigations of the pelvis of the kidney. The kidney function has not markedly improved and pus varies in amount, but edema has disappeared and the patient is feeling comparatively well. In the other case a recurrence in the opposite kidney followed a nephrotomy and drainage, and was either preceded or followed by a severe infection, attended by fever, chills and emaciation. This patient gradually improved under treatment carried out partly by myself and partly by her attending physician, has gained about thirty pounds in weight, has only slight frequency of urination and suffers no pain or discomfort in the region of the kidney. She came to see me to tell me that she was entirely well, and yet the *x*-ray plate shows four stones in the kidney.

8. *Conclusions.* Notwithstanding all that has been said or can be said, the conclusions of Cabot and Crabtree, in my opinion, are thoroughly sound. I trust that further experience will enable us to improve our results, but at the present time this is as far as we can go. They say: "On the basis of these cases we can only say to the patient that the risk of operation is small; that the danger of progressive destruction of the kidney by the stone, if it is left, is considerable; that it depends somewhat upon the age, undoubtedly somewhat upon the method of operation and the skill with which it is carried out, but clearly upon an entirely unknown factor—the liability or the ability of that particular kidney to form concretions.

Case I.—Mrs. R. C. A. was operated on June 7, 1907, for a right salpingitis and removal of the appendix.

About a month after leaving the hospital she had a chill, followed by fever and violent pain in left side. Shortly after she passed a stone and had relief of symptoms. In March, 1908, nine months later, she was complaining of pain in the lower abdomen, epigastrium and right shoulder, and also weakness of the bladder. Examination of urine showed pus and blood in the urine. In November, 1908, her urine had

become alkaline. In July, 1910, she aborted at three and a half months.

The urinary findings remained about the same except at times it became faintly acid and the amount of pus and blood varied. Several *x*-ray plates were negative for stone. From February, 1911, to February, 1912, she was comparatively comfortable. Catheterization of both ureters in May, 1912, showed a few pus cells from the left ureter and a great number from the right.

In September, 1915, she reported a continuation of pain in the back and bladder disturbance and an examination of the urine showed considerable pus and a few red cells. Cystoscopic examination showed congestion and edema of the base of the bladder. The flow from the right side was scanty in comparison with the left, and was cloudy. The right side showed pus and colon bacilli and the left side was negative. Following this four lavage treatments were given the right kidney. In October, 1915, leaded catheters were introduced into both ureters up to the pelvis of the kidney and an *x*-ray plate made before and after collargol injections.

X-ray plates showed a normal left kidney, and in the right kidney an enormous stone filling the pelvis and projecting into the calices. Five days later these findings were confirmed by a second *x*-ray.

In testing the kidney function the phenolsulphonephthalein was injected intravenously, and normally should show about 15 per cent. in fifteen minutes. In this test the right kidney showed only 2 per cent. and the left kidney 20 per cent. The destruction of the right kidney substance was well shown in the subsequent operation. Catheterization of the ureters gave the same findings as at the previous examination—the right kidney pus and colon bacilli, the left kidney negative.

On October 21, 1915, a right nephrectomy was done. The kidney was much enlarged and adherent. The ureter was divided as close to the bladder as possible.

Gross examination showed practically destruction of all func-

tionating structure, pelvis dilated and full of pus and stones. The major portion of the stone collection was in the pelvis, with numerous branches extending into calices. The stone was soft and showed characteristic fractures. The lining membrane of the pelvis was practically destroyed and the pelvic fat increased. Analysis of stones: calcium phosphate.

Twenty days later she complained of pain in left kidney and a large amount of pus in the urine. A cystoscopic examination made five weeks after operation showed the left kidney negative, but pus was seen oozing from the stump of the right ureter. A catheter was inserted and the stump was washed out with formaldehyde solution. Ten days later the left kidney showed on catheterization a few colon bacilli and epithelia, but no pus. The stump of the right ureter was still discharging pus. A plug of pus was washed out by irrigating with formaldehyde solution and 2 c.c. of pure carbolic acid injected. In July, 1916, she complained of a gnawing feeling in the left kidney and the bladder showed some pus. In January, 1917, the urine contained blood and had a slight uncomfortable feeling about the bladder, but no severe pain.

On February 13, 1917, a cystoscopic examination was made and the stump of the right ureter found obliterated. The left pelvis was dilated, the ureter was capacious and admitted a No. 11 Garceau catheter. The urine contained pus, blood and colon bacilli in abundance. The next day the *x*-ray plate showed two stones in the left kidney. The recurrence was demonstrated one year and four months after nephrectomy of the opposite kidney (Fig. 3). The stone in this kidney has never been removed. The pelvis has been irrigated at intervals with 15 per cent. collargol and she had been taking various urinary antiseptics. She remains in reasonably good health and has very little urinary disturbance. Her kidney function ranges around 50 per cent. in two hours.

Case II.—Mrs. L. B. was first seen November 23, 1916, at which time she complained of very frequent and burning urination. These symptoms commenced five years ago, with an attack of fever, pronounced typhoid. She at first had a stinging on urination and later frequent passage of a small amount of urine. After several years she began to have periods in which her symptoms became very much exaggerated and for the past two years has gotten steadily worse, until now she suffers constantly and is unable to go about or attend to her work. During the day she voids at intervals of from five to twenty minutes and is up and down all night. She now has marked tenesmus, but otherwise no pain except an aching over the sacral region.

Cystoscopic examination showed a marked inflammation of the base of the bladder, particularly associated with the right ureteral orifice; the left ureteral orifice was normal in appearance. Urine collected from the right kidney showed a large amount of pus; from the left kidney, negative. Cultures from the right kidney showed a growth of pure colon bacilli; from the left kidney; no growth. *X*-rays showed a branched stone in the right kidney, with considerable dilatation of the right pelvis. Capacity: right pelvis, 29 c.c.; left pelvis, 10 c.c. Function: right kidney, 10 per cent. in twenty minutes; left kidney, 25 per cent. in twenty minutes (Fig. 4).

She was operated on December 4, 1916, and the right kidney was found enlarged and adherent. It was bisected with wire and the stone easily located and removed. The cortex of the kidney was thinned, but the parenchyma appeared worth saving. The pelvis of the kidney was drained with a rubber drainage tube. Immediately following the operation the patient developed temperature, which continued irregularly for a period of several months.

On December 28, 1916, twenty-four days after operation, a cystoscopic examination with catheterization of ureters showed as follows: Right kidney (one operated upon): few pus cells and no bacteria. Left kidney (previously negative):

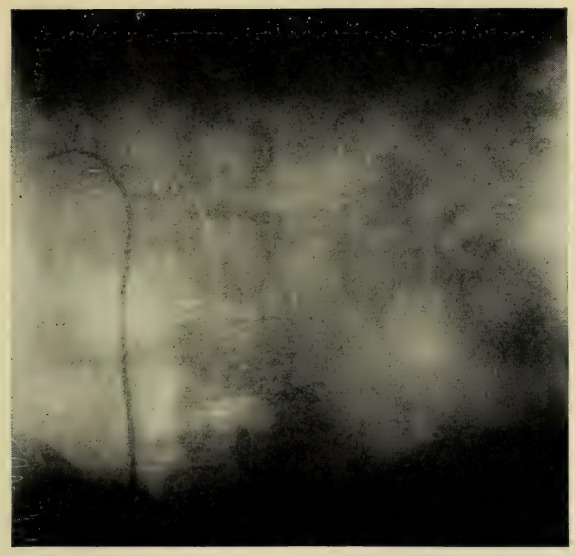

Fig. 1.—Branched stone in right kidney of Case I, previous to operation.

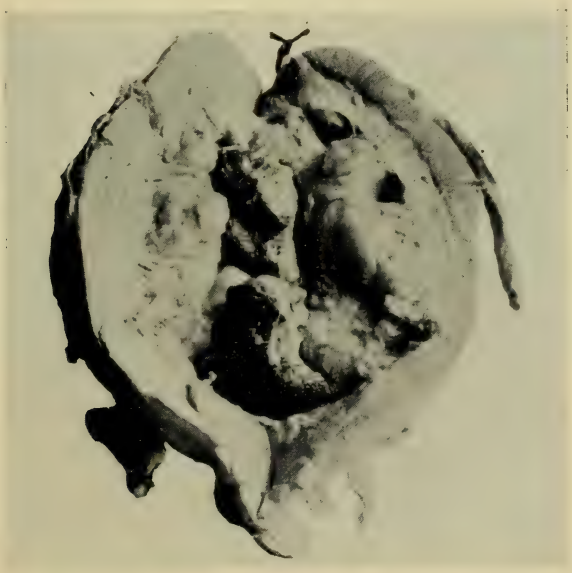

Fig. 2.—Right kidney removed from Case I.

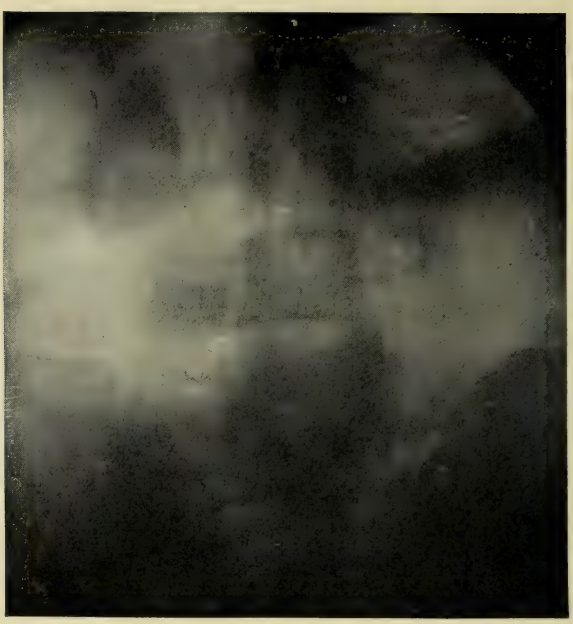

Fig. 3.—Recurrence of stone in opposite kidney after nephrectomy (Case I).

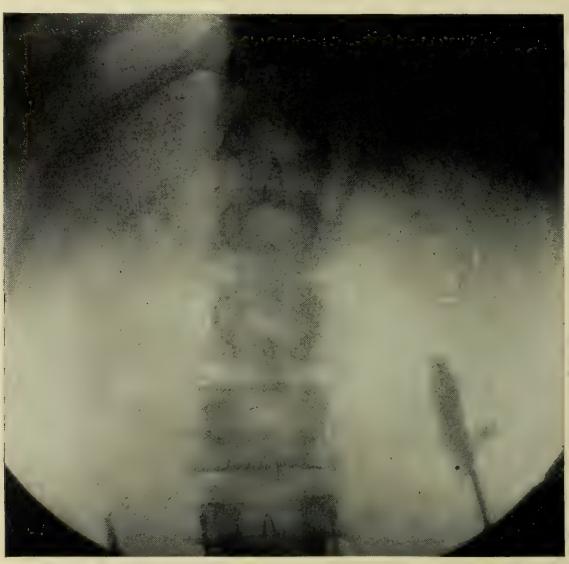

Fig. 4.—Branched stone in the right kidney with considerable dilatation of right pelvis (Case II).

Fig. 5.—Recurrence in the opposite kidney after a nephrotomy and drainage (Case II).

Fig. 6

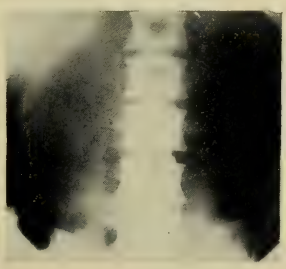

Fig. 7

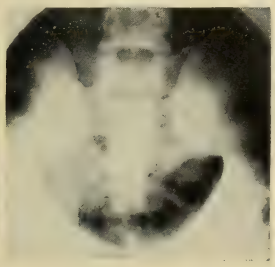

Fig. 8

Fig. 6.—Large stone in left pelvis of the left kidney. Right kidney and ureter and bladder clear (Case III).

Fig. 7.—Pelvis of left kidney clear (Case III).

Fig. 8.—Large concretion in the right ureter about four inches from the bladder (Case III).

Fig. 9.—Horseshoe kidney Left pelvis full of mushy material, right pelvis empty Small soft concretion in the left ureter about middle. Large stone in the right ureter about four inches from bladder (Case III).

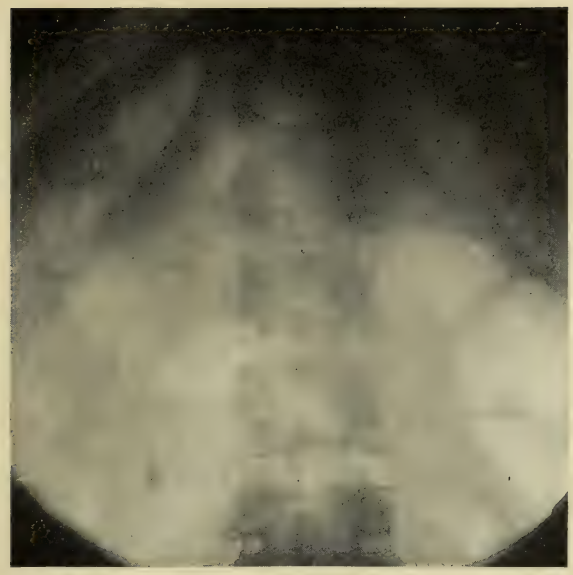

Fig. 10.—Recurrence of stone at site of previous removal (Case IV).

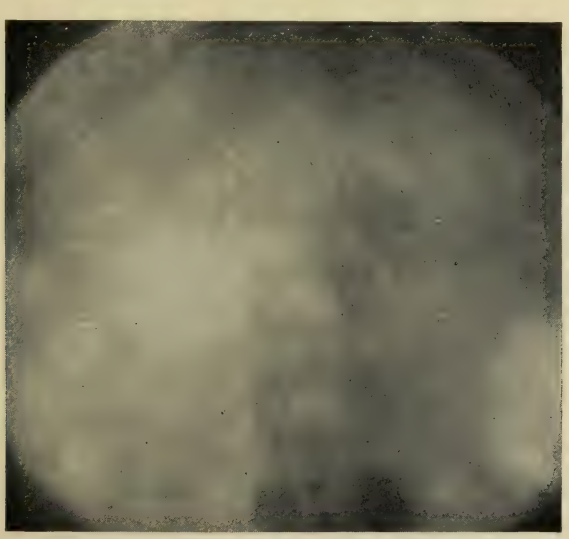

Fig. 11.—Second recurrence of stone at the site of the previous removal (Case IV).

large amount of pus and colon bacilli. The pelvis of the left kidney was subsequently irrigated without improvement, and in January, 1917, an *x*-ray plate showed four stones in the left kidney. These stones apparently formed in a month's time (Fig. 5).

Some interesting points in this case are as follows: On December 2, 1916, when the first examinations were made, the capacity of the right kidney as tested was 29 c.c. and of the left kidney 10 c.c., and the function of the right kidney 10 per cent. in twenty minutes, of the left 25 per cent. in twenty minutes. On January 30, 1917, a little over eight weeks later, the left pelvis had a capacity of 45 c.c. and the left kidney function was 9 per cent. in twenty minutes. The left kidney has not been operated upon. The patient was seen about two months ago and had fattened up about thirty pounds and complained of comparatively little urinary discomfort.

Case III.—J. A. G. was first seen November 8, 1916, when he stated that for years he had suffered with pain in the left kidney and marked bladder disturbance. On July 4, 1916, he was operated on at the Virginia Hospital and two stones removed from the left ureter. The *x*-rays at that time showed no stones on the right side. He left the hospital in August, and after going home had two attacks of kidney colic in the left side and three in the right side, and had had constant bladder disturbance and more or less constant pain in the back. Three weeks before, after a severe right-sided attack, he passed three stones, and when seen he was in the midst of an attack on the right side. One week later an *x*-ray plate showed the following: Large stone in the pelvis of the left kidney, two small stones in the right ureter about four inches below the kidney and an almond-sized stone in the bladder.

The stone in the bladder was crushed with forceps and removed through a cystoscope; a wax-tipped catheter was passed to a point of shadow in right ureter and returned with

scratches. In three subsequent cystoscopies the right ureter was dilated, the orifice stretched with forceps and glycerin injected and the stricture in the left ureter dilated also. After this the bladder symptoms cleared up entirely and he had no trouble until December 28, 1916, when he had an attack of pain in the left side again. The patient subsequently suffered considerable pain and in spite of every effort at medication the urine remained persistently alkaline. *X*-rays on February 1, 1917, showed right kidney, ureter and bladder all clear, and on the left side showed a large stone in the left pelvis which had definitely increased in size (Fig. 6).

On February 19, 1917, he was operated on. Through a nephrotomy opening a large collection of stones embedded in the granular material and enclosed in a membrane was removed from the pelvis of the kidney and the kidney pelvis drained. Analysis of stones: ammonium and magnesium phosphate.

While still in the hospital, about three weeks after the operation, he began to have pain in the right side and finally passed a soft stone. On March 30, 1917, the right kidney shut down completely; there was no bladder urine, but the left kidney was excreting freely from the drainage wound in the back. This condition continued and on April 3, 1917, *x*-rays showed left urinary tract clear (Fig. 7), but a concretion, the size of a peach-stone, was found plugging the right ureter about four inches from the bladder, which accounted for the anuria (Fig. 8). On the same day he was cystoscoped and a catheter stiffened with wire forced through the concretion, which was quite soft, and the tension relieved. This was done once after. On April 19 a right nephrotomy was done to relieve blockage of the kidney.

On May 2, 1917, the patient died and the following note was made of autopsy:

"Autopsy done and the entire urinary tract on both sides removed (Fig. 9). Kidneys and ureters very adherent. Lower poles of kidney connected across vertebral column by

a band of renal substance, about one and a quarter inches wide and one-eighth inch thick, making a complete horseshoe, the aorta lying *on top* of this band. Pelves and ureters entirely distinct throughout. Pelves entered kidneys anteriorly, blood supply going in behind and laterally. Left pelvis full of mushy, semimembranous material; right pelvis empty. Small, soft concretion in the left ureter about the middle. Large stone in right ureter about four inches from bladder; this stone was broken in two places and contained a hole which may have been the point at which the catheter passed through it. No stricture in the left ureter; the right ureter was much dilated. Base of the bladder inflamed. Nephrotomy openings in both kidneys. Left kidney markedly lobulated."

Case IV.—H. I. was first seen December 19, 1911, when he stated that four weeks previously, after wrestling, he was seized with an attack of pain in the left side and bloody urine. From this he gradually improved, until ten days before entering the hospital he strained himself holding a frightened horse, and this caused a recurrence of pain and bloody urine. The pain commenced near the backbone in the loin and ran down to the testicle, which retracted. The urine shows blood and the *x*-rays a small stone in the left ureter just below the pelvis of the kidney.

On December 29, 1911, the stone was removed by a pyelotomy, milking the stone back into the kidney pelvis. After this the patient made an uneventful recovery and remained well until November 2, 1914, when he complained of a dull, throbbing pain in the right iliac region, with frequent urination. He had been drinking a large quantity of water by direction.

Examination of the urine showed a few clumps of pus and a few blood cells and amorphous urates. The cystoscopic examination was negative except blood on both sides, which was probably traumatic, and a small amount of pus from both sides. *X*-ray plate was negative. On July 25, 1916, he

reported that he had had several attacks of pain on the left side and bloody urine.

X-ray plate showed a small stone at exact site of previous stone and same size (Fig. 10).

On July 31, 1916, a pyelotomy was done and the stone removed. Analysis of stone: magnesium phosphate. On February 1, 1917, he complained of a pain in the left side of the back and the urine showed blood.

On February 13, 1917, a cystoscopic examination was made and an obstruction encountered in the left ureter 25 cm. from the bladder. Wax-tipped catheter showed scratches. *X*-rays showed stone of the same size and location as formerly (Fig. 11).

The ureter was dilated and glycerin injected twice, but so far stone has not passed.

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DISCUSSION ON THE PAPERS OF DRS. HUNNER AND ROBINS

Dr. Bransford Lewis, St. Louis, Missouri.—I want to say a few words with reference to the paper of Dr. Hunner. To my mind the reality of ureteral stricture is well established and I do not believe any further argumentation is necessary except

in the case of some doubting Thomases. I have seen these cases in the clinic and have seen them postmortem, and I am firmly convinced.

There is one predicament in which we find ourselves once in a while in differentiating between the symptoms of stricture of the ureter and those of ureteral stone. I came across just recently a new sign that I think may be of some assistance in this connection. A patient was sent to me from Illinois, who gave very typical symptoms of ureteral colic. A radiogram showed a shadow looking like a stone apparently in the line of the left ureter. A second radiogram taken with a radiograph catheter in the ureter showed the same shadow and apparently in contact with the catheter, seeming to confirm the presence of ureteral stone. But failing to feel any obstruction at that point in the ureter I was unconvinced. I thereupon inserted a flexible ureteral forceps into the ureter, past the location under question, and had another radiogram taken. This was stiffer than the catheter had been, and therefore straightened out the ureter; the simulated stone shadow was shown three-quarters of an inch from the catheter's shadow, thus plainly demonstrating it to be the shadow of a phlebolith and not a stone. The sign was explicit and irrefutable.

Dr. Mason.—You mean to omit the catheter?

Dr. Lewis.—The catheter being flexible and softer went around and happened to be exactly in apposition to the shadow of the phlebolith.

It might possibly prove misleading enough to get one to operate in a similar case.

I have at the hospital at the present time a patient, about seven months advanced in pregnancy, who has acute pyonephrosis. She has a temperature of 104° and all the evidences of urinary sepsis. She has been treated by various means that have not afforded any relief until we got a catheter up in the left ureter, which brought good urine, but on the right side we drew out about two ounces of purulent urine. Immediately the temperature was reduced from 104°, with recurring chills and evidences of sepsis down to 98.6°. I left the ureteral catheter up there to drain the kidney continuously, and twice a day we washed out the pelvis of the kidney. That was not a ureteral stricture, but an obstruction incidental to the pregnancy and puerperal pyelitis like that referred to by Dr. Hunner. An interesting feature is that it required the retained catheter in

there to make the desired effect, because the first time I drew off the urine from the right kidney pelvis she was all right for a day and the next day she had a recurrence of the fever. I introduced the ureteral catheter and left it up there until I left to come to this meeting, and at that time she was doing very well.

Dr. James E. Thompson, Galveston, Texas.—There are many aspects of this question that are intensely interesting, but there is only one question that I shall ask Dr. Hunner to answer. We are quite convinced as to the immediate efficiency of dilatation of ureteral strictures, but have the cases been observed long enough to give us a true indication as to the permanent effect? Do these strictures behave like those of the urethra and will the dilatation have to be repeated from time to time to keep them open?

As to Dr. Robins's paper, I have long been interested in the subject from a physiological point of view. One knows perfectly well that these unfortunate patients pass through a period of life when the stones form quite frequently and rapidly. I have in the University of Texas museum a collection of over sixty uric acid calculi (gravel) passed spontaneously by the same individual during a period of about six years. The stones vary in size from a hemp-seed to a pea, and two or three are elongated in shape, having evidently been molded by the ureter. The last stone to pass out from the ureter into the bladder was unable to pass into the urethra and formed a fair-sized calculus in the bladder, for which I operated. For a year and a half subsequent to the passage of this stone along the ureter calculus formation had ceased in the kidneys. I have another patient under observation from whose bladder I removed nine stones of varying sizes. From the history they were all formed in the left kidney and passed down the left ureter into the bladder. They were passed during a period of two years, and the intervals were irregular. For almost a year before the operation of cystotomy there was no evidence of calculus formation. This case was interesting from another point of view in that the *x*-ray shadow of the stones was somewhat blurred, but the pelvic veins were full of phleboliths, which cast sharp, distinct shadows. These were mistaken for those of stones before operation. An *x*-ray picture taken after the cystotomy showed the shadows of the phleboliths undisturbed while the blurred shadow of the mass of stones was missing. This patient has

a high uric acid output which we are unable to reduce by diet. It is necessary for us to study these cases from a physiological stand-point and to call to our aid a physiological chemist. We know that under ordinary circumstances only a small proportion of the nitrogen set free by the processes of metabolism passes out in the urine as uric acid, the great bulk of it passing out as urea, which is very soluble. Uric acid, on the contrary, is less soluble and is apt to be deposited as crystals in the tubules and pelvis of the kidney. Uric acid belongs to a group of bodies which are known as the purin bodies. In the same group are xanthin and hypoxanthin, which occur in meat extracts and tissues rich in nucleoprotein, such as sweetbreads, liver, the roe of fishes and gamy meats; also caffein and theobromin and certain vegetables, such as the leguminous plants and asparagus, are rich in purin bodies. The purin bodies appearing in the urine (uric acid, xanthin, etc.) are derived from two sources, viz.: (1) from the food, and (2) from the tissues. We speak of those derived from food as of exogenous origin, because they have never been built up into the protoplasm of the cells of the body but have simply passed from the stomach and intestinal canal into the portal veins and thence into the general circulation, to be eliminated eventually by the kidney epithelium. Those that are derived from the tissues we speak of as of endogenous origin, because they result from the combustion of living protoplasm. It is possible to eliminate from the diet all purin bodies by confining ourselves strictly to a diet of milk and eggs and vegetables free from purin. Under these circumstances the amount of endogenous purin excreted daily becomes a fairly constant quantity, depending on the weight of the muscles. It is liable to variations, being increased by any cause which induces more combustion, such as exercise or fevers.

It will be obvious that under diets rich in purin bodies the quantity of uric acid will be increased in the urine, and if conditions are suitable that crystalline deposits will be liable to form in the urinary passages, and, further, it is obvious that we can, by regulating the diet, exclude from the urine all purin bodies of exogenous formation and reduce the output of uric acid to a minimum. We know still further that the liver has the power of transforming these bodies into the more soluble urea and that the activity of this organ is a necessary adjunct to the reduction of uric acid.

Unfortunately our clinical cases do not respond satisfactorily to dieting. I have never succeeded in stopping the tendency to gravel formation by the most rigid diet. It appears as if these individuals pass through a period of susceptibility to stone formation which is more or less independent of the amount of uric acid in the urine, and that after the lapse of a certain time this period of susceptibility disappears.

Dr. Robert L. Payne, Norfolk, Virginia.—The last word has not been said on this subject, and it is going to be a long time before it is said. From the two papers that have been presented there are two facts that stand out very forcibly, one of which is infection and the other obstruction. The fact is more or less established that the kidney is a great bacterial filter, and given those cases of infection in which stones have been removed, with the rich blood supply that the kidney affords, surely it must be the obstruction laid down on top of that infection, or with it, that tends to the reformation of stones in these cases. I have seen, unfortunately, a good many examples, a very recent one as follows: A patient had a stone removed in Baltimore from the ureter at a position opposite the second lumbar vertebra. Three months later, in my home town, he had a severe attack of kidney colic; he fell into my hands; an *x*-ray plate was taken and was found negative. The ureteral catheter disclosed a stricture which was dilated and the symptoms disappeared. The patent went then to a lung specialist and was under his care for three months; he had two severe attacks of colic, with a tremendous return of pus in the urine. An *x*-ray plate showed stone formation just above the stricture located by me in the second instance.

Now, traumatism to the kidney plus infection is a marked predisposing factor to stone formation, and I have at home microscopic sections of two experimental kidneys in which uranium nephritis was produced, with infection laid down upon that by means of colon bacillus, and in two instances stone formation occurred. There were forty dogs in the same series that had uranium nephritis in which infection was not laid down upon uranium traumatism and in which stone formation did not occur. If there are any doubting Thomases relative to Dr. Hunner's ideas about ureteral stricture, the slides shown by Dr. Robins on the screen of the kidney will dispel that doubt.

It seems to me, in almost every instance of the 4 cases reported

by Dr. Robins, that obstruction was at the bottom of the return of the symptoms and that one slide showed obstruction in the lower ureter in both instances, and especially in the side where the stone recurred in the ureter. There is one lesson to be learned from this little argument and that is this: we are not paying enough attention postoperatively to surgery on the kidney, and especially our surgery on the ureter, and for the past few years I have made an invariable rule to follow every kidney and ureteral case afterward, with the view of establishing absolute and free drainage in the ureter, especially in ureteral stones removed surgically and surgery of a plastic type done on the ureter. Stricture formation is liable to occur, and unless we watch for this carefully and pass ureteral catheters and formulate some rule as to how many pus cells there are in a field we cannot judge as to whether a kidney is draining properly. If we dilate the ureters after we remove stones from them, or after we have operated upon them for other purposes, in many instances I think we can forestall the continuation of the infection and second the stone formation.

Dr. W. R. Jackson, Mobile, Alabama.—I should like to say a few words about the subject: first to thank Dr. Hunner for his explicit, philosophical and scientific explanation of ureteral stone. Heretofore I had always believed, and I believe most of the text-books teach, that ureteral stones come originally from the kidney. They are exact in formation. Prerequisite conditions for the formation of any stones are (1) a nucleus; (2) denudation of the epithelium; (3) stasis of urine. These are the essential etiological factors in the formation of stones. Stasis of urine usually takes place in the pelvis of the kidney. I never thought before that a stricture of sufficient degree would be capable of producing stasis of the urine in its exit from the ureter to form a stone or to give rise to the nucleus of a stone *in situ* at the stricture formation. I have always thought the force of urine carried it out and it could not linger long enough to produce a stone at the stricture point. But another view-point can be taken. We now know that stone can produce strictures. The doctor's claim may be *post hoc* instead of *propter hoc*. A stone will also produce a stricture of the ureter. The essential and underlying element of these strictures is infection with submucous infiltration, and fixation of the stone is followed by ulceration, particularly if the stone remains there for any considerable

length of time, and as a result of this ulceration we have cicatricial contraction and stone formation. Strictures form stones, and *vice versa*, like infection; hemorrhage favors infection and infection favors hemorrhage.

The recurrence of stone is due to the same essential factor that causes its primary formation, and the rapidity and certainty incident to the recurrent formation of stone depends upon the extent or degree of the predisposition to the formation of stone. As Dr. Payne said, uric acid is the essential factor or the nucleus in the formation of stone. Further, infection is necessary to give rise to this cement substance from the blood. Therefore, you may calculate on your protein element for stone formation of any kind. Another thing: may we not overlook stones when they are already present even by an *x*-ray examination? The question is whether the *x*-rays will show up a stricture when the waxed-tipped ureteral catheter shows stone scratches upon it. We cannot always say. From the viewpoint of older surgeons I should say we have stones which originate in the kidney, and whenever a stone encroaches upon the sensory filaments of the mucosa of the ureter, in any part of its course, we have ureteral spasm which we term ureteral colic (renal colic). While it is in the kidney, remaining dormant, it produces no symptoms except those of soreness or a heavy pain in the lumbar region and frequency of urination.

As to therapeutic measures, the stretching of a stricture does not cure it; it does not matter where the stricture is located, whether in the ureter, the urethra, the esophagus, the intestinal tract or what not. It comes back again.

Dr. Edward H. Ochsner, Chicago.—It seems to me there is one etiological factor that has not been mentioned here today and that is the question of traumatism. One Fellow mentioned two points in the formation of stones in the ureters and kidneys, and these were infection and stricture. I believe there is a third, namely, traumatism. I think that traumatisms of the kidney occur much more frequently than we have heretofore thought. A sudden traumatism which causes hemorrhage at the pelvis of the kidney is not an uncommon starting-point for a stone.

There is another thought which comes to me in connection with this subject, and it is this: I think we are too anxious to make a correct diagnosis. We are more concerned about the correctness of our diagnosis than we are about the welfare

of our patient. I think we sometimes fear undue criticism by not going into a thorough study of our cases, so that there is a possible loophole for criticism. What I mean is this: in the study of these pelvic infections we should never introduce an instrument on the side where there are no symptoms, providing there is infection on the other side.

I do not think we ought to catheterize the ureter of an apparently sound kidney—a kidney from which there are no symptoms. I do not believe that twenty years ago there were as many recurrences of stones following kidney operations nor were there as many secondarily involved kidneys as there are today, and one reason for this is that at that time there was very little ureteral catheterization practised. It is impossible to sterilize an infected bladder—it is folly to attempt it, as it cannot be done. I believe it is more important to relieve the patient of his symptoms and his ailments than to make an absolutely correct detailed diagnosis.

Dr. Joseph Ransohoff, Cincinnati, Ohio.—I should like to ask Dr. Hunner whether he believes infection is absolutely necessary for the formation of a stone or whether or not an abscess is necessary for the existence of a stone? That seems to be the general belief because some authority, like Epstein, has said so. We all follow shibboleths, and this is one of them. For my own part I believe very often these stones are formed without any organic basis and without any infection. I have a man who has gone on for six to eight months passing a little gravel. This gravel is about the size of pinheads, but rarely as large as a pea. In the interim the urine is absolutely normal. His ureters have not been catheterized. After he had passed some of these stones we took an x-ray picture and could find nothing in the kidney. That man continues to manufacture stones. He has no infection and I do not believe there is an organic cause for these little stones.

I am reminded in this connection of the question, Why is it that in the Mammoth Cave we have stalagmites and stalactites when there is no organic basis for infection?

Dr. Hunner (closing on his part).—First, I wish to express my appreciation of Dr. Robins's classical paper. He has dealt with an extremely important subject, and I think the lessons we can draw from his paper are, in the first place, that it is

extremely important, before we operate on a kidney for anything, particularly for a stone of the kidney, to know the condition of the other kidney, and to know how far we can go in our radical or conservative methods with the one we are going to operate on. My personal reference is, when I find a stone in the kidney the seat of a fibrosis or lipomatous pelvis, or if it is extremely dilated and has a thin cortex, to remove that kidney, providing the other kidney shows every evidence of being healthy. If we have not investigated the other kidney we are working in the dark and would best be careful about the one we are working on, even though there is a big chance for the reformation of stones in such a case.

The second point I got from Dr. Robins's paper, and that has been emphasized by Dr. Payne, is that we should follow up these cases better and see if we cannot keep the obstruction from returning, if infection is being continued, because of stricture or obstruction in the lower ureter, and we should wash out the kidneys with antiseptic solution and get rid of the pyelitis. That is not necessary immediately after operation any more than it would be in a bladder case. We remove stones from the bladder and in a few months the urine becomes normal and infection clears up because we have taken out the foreign body. The same thing happens in the kidney, but many of them do not clear up spontaneously, and in these we adopt a method of drainage to clean them up.

Dr. Thompson asked about the permanence of the dilatation and what the ultimate fate in these ureteral stricture cases is, and Dr. Jackson answered that question in part by saying that we do not get rid of the stricture if we stretch it. In other words, we do not cure stricture. That is very true; at least, I suppose it is true, because if a patient gets well I do not get a chance to examine her to see what has become of the stricture. I have no idea that a stricture disappears after stretching, but the results of the stricture disappear, and that is what we are after. The symptoms clear up and the pelvis of a kidney holding 40 c.c. when we first begin treatment may, after a few dilatations of the stricture, contract so that it holds 8 to 10 c.c., and when the symptoms all disappear I consider that satisfactory work. Unfortunately not all of them are that satisfactory, particularly in the type of cases that do not have dilatation of the pelvis of the kidney above the stricture, and that I will not discuss at length here because you will find the ground covered in my

article on "Ureteral Stricture" in the *Transactions* of last year. In that type of case when there is a contracted pelvis of the kidney rather than a dilated one above the stricture the results are not so satisfactory, and we do not claim we cure them. Some of these cases still complain. On the whole, however, the method is extremely satisfactory.

As to those cases that have evidences of ulcer at the stricture site with bleeding I have generally diagnosed them as stone cases until the investigation showed the absence of stone and presence of stricture. The ulcer eventually clears up under dilatation treatment and the bleeding ceases.

In regard to trauma by the wax bulb in dilating the stricture, this instrument brings about a new circulation and the ulcer heals, as shown by the disappearance of the blood from the urine.

I was much interested in Dr. Lewis's new sign of using a stiff metal dilator in getting his *x*-rays. In a way we can imitate that simply by leaving the stylet in our catheter, but it is bound to take the normal shape of the ureter and would not be as effective as a stiff metal dilator.

Dr. Lewis.—The stylet was used in this case.

Dr. Hunner.—I thought you used the ordinary catheter. In my paper of last year I called attention to the frequency of occurrence of phleboliths in the broad ligament region in the uterine vein where the ureter crosses under and the danger of getting a superimposed picture, and have recommended the use of the bifocal method of *x*-ray photography to bring up the third dimension.

Dr. Ochsner warns against the possibility of infecting the other kidney. I sounded that note of warning twenty years ago, but I must say that my continued work along this line has rather convinced me that that danger is more theoretical than real. I think all urologists who follow their cases very carefully and follow them up by bacteriological examinations have no reason to think there is any great danger of infecting the opposite kidney in going through an infected bladder. I try to avoid it where I do not think it is absolutely essential. I catheterize the infected side and leave the catheter in if I want to make a functional test and gather urine through the bladder from the other side. As a matter of experience I have yet to see the case I have so infected by catheterizing through an infected bladder.

With reference to the question of Dr. Ransohoff as to whether

infection is necessary for the formation of stones, I will say that in the majority of my cases of stone in the ureter they were not infected—at least the urine was not infected. They may originally have had a nidus of infection and the urine may have cleared up after the stone had formed. This is contrary to our usual experience. There is a foreign body, and if there is infection we would expect it to remain as the result of the foreign body. But most of these cases are associated with non-infected urine, and I do not believe infection is necessary in the formation of stone.

As to the question of an organic basis for stone, that depends upon what we are going to use as a definition of organic basis. I cannot imagine a stone forming out of a clear sky or in the ether, as it were, not being able to define what we mean by ether. In my judgment a stone must have some kind of a foundation. In the case of ureteral stricture I think it is a little oozing of blood from the ulcer of the stricture, or if there is no ulcer it is rather a hypersecretion of mucin, and if it is not mucin it may be serum or mucoid material. There must be some organic basis, and in the case Dr. Ransohoff mentioned, in which the patient manufactured stones, I will say that I have an identical case in a woman who has no infection in the urine but every now and then she comes in with a bottle of uric acid stones of various sizes which she has been passing. I think they come from some trouble in the renal tubules: that there is some catarrhal condition or hypersecretion which accounts for the deposit of these uric acid particles in the formation of the stone.

Dr. Ransohoff.—Are there any tubules in the Mammoth Cave?

Dr. Hunner.—You get your matrix of earth to start with. The first calcareous deposit has taken place on the wall of the cave on the earth itself and the others would seem to be increments on top of that.

Dr. Robins (closing).—In reference to the relation of ureteral stricture to the formation of stones, I will say that in preparing this paper I reviewed the literature, but did not quote anything unless it had a direct bearing on the cases I reported. Dr. Fowler, of Denver, read a paper recently in which he ascribes stone formation to two conditions found in the urinary tract. One of them is ureteral stricture and the other malposition of the kidney. He described a new operation for fixation of the

kidney. In this paper he referred to the work of Dr. Hunner on ureteral stricture.

In one case particularly, which was under observation for ten years, the woman had stones to form in one kidney while the other kidney was absolutely free. We determined that point. She had infection for a long time but repeated *x*-ray examinations failed to reveal the stones until the last examination, when we discovered them. That kidney was removed. She then had stones to form in the other kidney. If obstruction had anything to do with it, why did not they form before? I do not think obstruction in the ureter had anything to do with this case. I believe there are two principal factors in the formation of stones as stated by Brewer. One of them is infection undoubtedly, and of course we may have infection plus other things. In the other it is due to an error of metabolism, whatever that may be. In those cases we do not know really why the stones form. We all have seen cases of gravel in which small concretions or sand lodge temporarily and then pass. This may go on for years, with attacks at frequent intervals, without the formation of a definite stone. These people undoubtedly have some error of metabolism. We doubtless have a combination of the two conditions in some cases.

I have reported these cases because I think they are examples of a large class of cases that have been overlooked. Nobody has been looking for recurrences of stone in the kidney, and consequently we have not seen them.

RETROGRADE PERISTALSIS IN THE SECOND AND THIRD PORTIONS OF THE DILATED DUODENUM

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and

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From a strictly surgical stand-point the first portion of the duodenum ranks far ahead of the second and third portions, but it is quite possible that we have had our attention drawn to the former by its frequent surgical lesions and have neglected the interesting possibilities of the latter.

Believing there is really "nothing new under the sun," I am attempting a brief description of the phenomenon, which serves for a title to this paper, for the purpose of creating discussion, and, if possible, developing an interest among us sufficient to keep us on the lookout for further instances. My colleague, Dr. Smith, a man of wide experience in laboratory diagnosis, has never before observed it. I have discussed the matter with a goodly number of roentgenologists, none of whom has noted it, and I have been unable to find it recorded in the literature of the subject. It is only fair to say, however, that my search of the literature has been limited by my facilities for such investigation, and it is quite possible I have overlooked it.

Regardless of all this negative evidence it is inconceivable that there can be but one case in the world suffering as did mine, and it is my belief that if a sufficient number of similar

cases could be studied we should add very materially to our evidence that lesions of the ileocecal segment are often responsible for symptoms referable to the epigastric region.

Adventitious bands and other causes of kinks throughout the intestinal tract have interested surgeons and internists alike since the development of the fluoroscope to the point where observations through it became dependable.

Either dilatation or hypertrophy must occur in the portion of the gut proximal to long-continued mechanical obstruction of any considerable degree, and the duodenum is no exception to this rule. That hypertrophy of the duodenum may follow kinking of the gut at the duodenojejunal junction is a generally accepted fact, although it is usually spoken of as chronic dilatation. In my opinion this latter term is incorrectly used, for I believe it to be a physiological impossibility for the duodenum to remain chronically dilated. However, I shall make use of the term again, for, whether correct or incorrect, it is familiar to us all.

I have long held the theory that the duodenum may become dilated or hypertrophied when no obstruction can be demonstrated at the duodenojejunal junction, and in seeking to prove or disprove this theory we have made it a routine in our clinic to make careful and repeated fluoroscopic observations of this portion of the intestinal tract in all cases which are subjected to roentgen-ray study after a barium meal.

In 1 such case one of us (Smith) observed the phenomenon which furnished the title for this brief paper. The experience was so unique that he summoned me to the *x*-ray department, where we both watched it occur over and over again. The following day, when the patient returned for the eighteen- and twenty-four-hour plates of the colon, she was given another pint of barium pap, and again we observed, for a period of about an hour, the same rhythmical filling and regurgitation which Dr. Smith has described as follows:

"Chronic dilatation and varying degrees of obstruction of the third portion of the duodenum has been clinically demonstrable for several years. But just why reverse peristalsis does not occur in this portion of the gut when it becomes obstructed, similar to the portion proximal to an obstruction in any other point in the gut tract has not been so readily apparent. A certain few of these cases of chronically dilated and obstructed duodeni have been under my observation, and it is in just such cases as these that the opportunity has arisen to study antiperistalsis in the second and third portions.

"During the fluoroscopic examination of the gastro-intestinal tract, after the administration of cornstarch pap and barium sulphate, the stomach was observed as a hypotonic organ, situated unusually low in the abdominal cavity, with very shallow and slow peristaltic action. The pyloric ring at first appeared apparently normal, allowing the duodenal cap to be visualized with considerable clearness. The first few onward spurts of the barium pap passed through the second and third portions of the duodenal tract into the adjacent jejunum, but it was evident that the duodenum was not properly clearing itself. The duodenal contents began to stagnate and in several more minutes its lumen was filled to what one considers a normal width of the duodenal shadow. During the next several minutes the duodenal musculature was seen to undergo forceful peristalsis, each time discharging a small amount of the barium pap into the jejunum, but was practically unable to clear its then dilated lumen. While observing this vigorous peristaltic action a reverse peristalsis was suddenly instigated and the entire contents of the dilated duodenum was discharged *en masse* backward, past the pyloric ring, into the stomach, somewhat after the manner in which the colon by its mass action clears itself in a rhythmic action about once in twenty-four hours—the familiar mass peristalsis of Haudek. During this process the patient complained of the same pain from

which she had suffered for the past four years. Finally the pain subsided, and again in four or five minutes the same process described above began again, accompanied by the same distressing pain. Each time a certain amount of the barium pap was discharged into the jejunum. Apparently this does not happen in the recumbent position, for a long time ago this patient found relief for this pain by lying down with the feet and hips elevated. At any rate, when she was placed upon the horizontal radiographic table, for the purpose of making a few roentgenograms for records, the duodenum emptied itself, the jejunum filled, and when the six-hour plate was taken the stomach was completely empty. It is again significant that the terminal portion of the ileum did not empty until the twentieth hour, that the cecum was dilated and displaced in the pelvis, that the transverse colon was deeply situated in the pelvis, and finally that the stasis was limited to the right lower quadrant of the abdominal cavity. All of these pathological changes have been noted as occurring in conjunction with chronically dilated and obstructed duodeni."

I trust you will pardon a somewhat lengthy quotation from her history sheet, but she belongs to a class which furnishes some of the most perplexing problems I am called upon to solve, and I suspect the same is true with surgeons everywhere.

The patient, Miss J. D., aged twenty-nine years, a teacher in the public schools, was referred to us by her sister, who is a trained nurse. The family history was unimportant. She was born in Africa, where her father was then engaged in missionary work, but came to this country when only a child. Aside from the usual diseases of childhood, from which she made good recoveries, her history was uneventful until her menses appeared at the age of fifteen. Since then she has suffered more or less severely from dysmenorrhea. About ten years ago she had an attack of "slow fever" and was in bed two months. Since then she has not been strong but

succeeded in finishing her college course and in holding her position as a teacher.

Some four or five years ago she began to suffer from somewhat vague abdominal pain, which, in a rather indefinite manner, she associated with the taking of food. She had constipation to a degree requiring the constant use of laxatives, had suffered much from headache, which her oculist assured her was due to her general condition, and had lost ten pounds in weight during the preceding school year. Her appetite was capricious and she frequently deprived herself of lunch because by so doing she could continue the afternoon session of school with less discomfort. Her physical strength was declining rapidly, and it had become extremely difficult for her to continue her work. She was exceedingly nervous, discouraged and even alarmed about her condition, fearing that she would soon be obliged to give up her work altogether. She had been examined and told that she had a retroverted uterus, which was responsible for her symptoms, and she came to us with the idea of undergoing an operation for this condition.

Physical examination showed a pale, anemic little woman, whose stature suggested a child of fifteen rather than a woman of twenty-nine. She was five feet two inches tall and weighed eighty-four pounds, which is some thirty pounds below the normal. Head, neck and chest were negative. By percussion and palpation of the abdominal wall it was quite apparent that she had a marked, general enteroptosis; the right kidney was easily palpable and freely movable; there was apparently considerable tenderness over the whole right lower quadrant. Vaginal examination was unsatisfactory, but the cervix could be felt with the finger tip one inch within the introitus; by rectum an elongated, conical cervix could be made out, and the fundus was retroverted, with a considerable degree of prolapse. Blood examination was negative, except that the hemoglobin was only 70. The urine was negative.

If my word-picture has been reasonably clear the type of case will be instantly recognized and my reluctance to subject her to operation will be readily understood.

Three points in the history impressed me: the obscure, but none the less unmistakable, sense of discomfort after meals, the obstinate constipation and the severe and persistent headaches, which were clearly of a toxic origin. After further consideration I advised radiographic examination of the alimentary tract, and told her that if mechanical interference with the fecal current could be demonstrated surgical intervention should be contemplated.

After receiving Dr. Smith's roentgen-ray diagnosis of chronic appendicitis and marked stasis in the terminal ileum I felt justified in advising laparotomy.

Accordingly, on July 25, we opened the abdomen under gas-ether anesthesia. The gall-bladder and pylorus were normal; no obstruction could be demonstrated at the duodenojejunal junction; the spleen and left kidney were normal; the right kidney was normal to palpation but had a somewhat abnormal range of excursion. The cecum was low in the pelvis but was freely movable; the terminal three or four inches of the ileum was lying over the pelvic brim and tightly bound down by the so-called ileopelvic bands; the appendix was adherent to the cecum for about two inches of the proximal portion, the tip being free.

The ileum was liberated, the appendix removed, the utero-sacral ligament was shortened and a round ligament operation done for the retroversion. We had previously dilated the cervix, amputation not appearing necessary.

Her operative recovery was uneventful and her subsequent history quite satisfactory. She returned to her school work in September, as she is forced to be self-sustaining, and is undoubtedly working beyond her strength. In spite of this handicap she has gained fifteen pounds in weight, her headaches have practically disappeared and her epigastric pain and constipation have been entirely relieved. She still

has a moderate amount of menstrual discomfort, but it is not sufficient to interfere with her duties.

The results in this case cannot, by any stretch of the imagination, be called brilliant, but it is evident that Nature never intended this woman for the robust type, and her improvement has been so marked that she is thoroughly satisfied. The most interesting feature of the subsequent history is that a fluoroscopic examination made about three months later showed perfectly normal emptying of the duodenum.

We propose to subject her to fluoroscopic examinations from time to time for the next year or two for the purpose of determining whether or not the cessation of the regurgitation is permanent.

In conclusion I will state again that I am reporting this case for the purpose of recording a phenomenon which I have never seen before, which I have never seen described and which, to me, was exceedingly interesting.

I believe the epigastric discomfort she complained of was due to the violent muscular efforts of the duodenum to empty itself and that the actual pathology was in the ileocecal segment of the gut. I further believe that continued study of similar cases, carefully checking up the fluoroscopic observations with the operative findings, will reveal a constantly increasing number of dilated or hypertrophied duodeni, where the only actual obstruction will be found in the terminal ileum or in the colon.

THE MANAGEMENT OF SUBPARIETAL INJURIES OF THE KIDNEY: WITH REPORT OF THREE CASES

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These injuries vary much in severity, and may consist of contusions, lacerations or rupture of the kidney. They may be simple, that is, confined to the kidney alone, or complicated by injury to the bloodvessels, ureter, peritoneum or other adjacent structures, or to any other tissue or structure of the body. As kidney injuries are due to such a variety of traumata, the associated lesions are not subject to classification but must be considered individually.

No large collection of kidney injuries is likely to occur in the practice of any one surgeon, but the literature contains many isolated case reports, numerous small series of cases and several tabulated collections of large numbers of cases, with analysis of the results obtained by various methods of treatment.

For the purpose of this paper it has not been deemed necessary to collect all the reported cases, as this has been done repeatedly, and the figures have been brought up to date as recently as October, 1916, by Bugbee.[1]

We have preferred to discuss the subject from materials before us, modified by more recent advances in surgical technic, kidney functioning tests and safer anesthesia.

Hematuria, pain and tenderness over the kidney, the development of a tumor about the kidney, together with

varying degrees of shock and collapse, following a history of trauma to the loin, indicate an injury of gravity to the kidney.

As many cases are recorded in which shock and severe pain were absent or delayed, we should be carefully on guard when trauma is followed by hematuria, and should keep such a patient perfectly quiet and under constant observation until the nature of the injury is determined.

Expectant treatment, early exploration and late operative treatment have been the plans pursued in dealing with these injuries.

Expectant Treatment. Michelson,[2] from Riga Hospital, for twenty years preceding 1911, reports 30 cases of rupture of the kidney, all of which were treated expectantly, with only 3 deaths, or 10 per cent., and in all the fatal cases there were severe complicating injuries: (1) compound fracture of the leg, fracture of the forearm, multiple fracture of ribs; (2) rupture of liver, multiple fracture of ribs; (3) fracture of forearm, multiple fracture of ribs, pneumonia.

Ponomareff,[3] from Municipal Hospital Obishoff, St. Petersburg, 1898–1912, reports 57 cases of ruptured kidney, with the following results: Three cases left the hospital untreated and were lost sight of; 3 died as a result of complicating injuries; 43 recovered under expectant treatment; 8 recovered under operative treatment.

From these statistics, expectant or non-operative treatment in the uncomplicated cases would seem to be without mortality. Watson,[4] however, reports a mortality of 27 per cent. under expectant treatment in 237 collected cases.

The statistics of Michelson and Ponomareff, which are widely quoted, have given "expectant treatment" a status to which, in my judgment, it is not entitled. Furthermore, of the large number of cases reported by them as ruptured kidneys, unconfirmed by operation or autopsy, some must have been injuries of much less severity.

Concerning this plan of treatment, Neilson[5] says: "So-called expectant treatment is permissible only in cases in

Fig. 1.—Rupture of the left kidney. (Case III.)

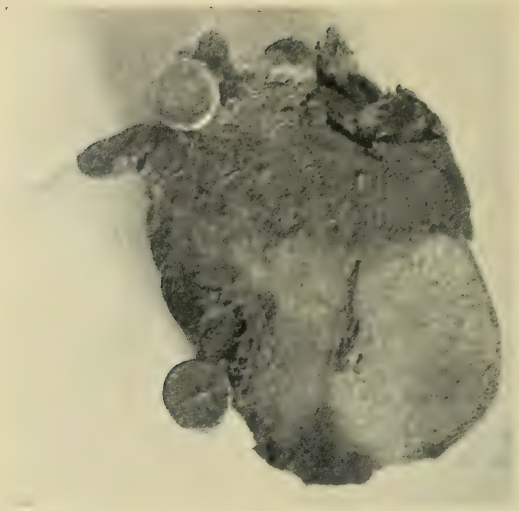

Fig. 2.—Section through ruptured kidney (Case III), showing line of demarcation and areas of necrosis.

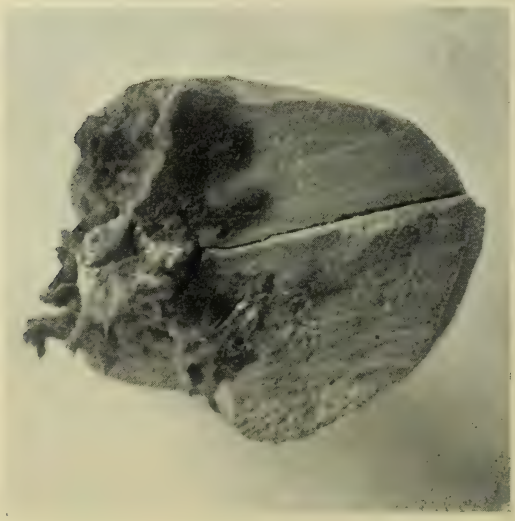

Fig. 3.—Section through ruptured kidney (Case III), showing line of demarcation and areas of necrosis.

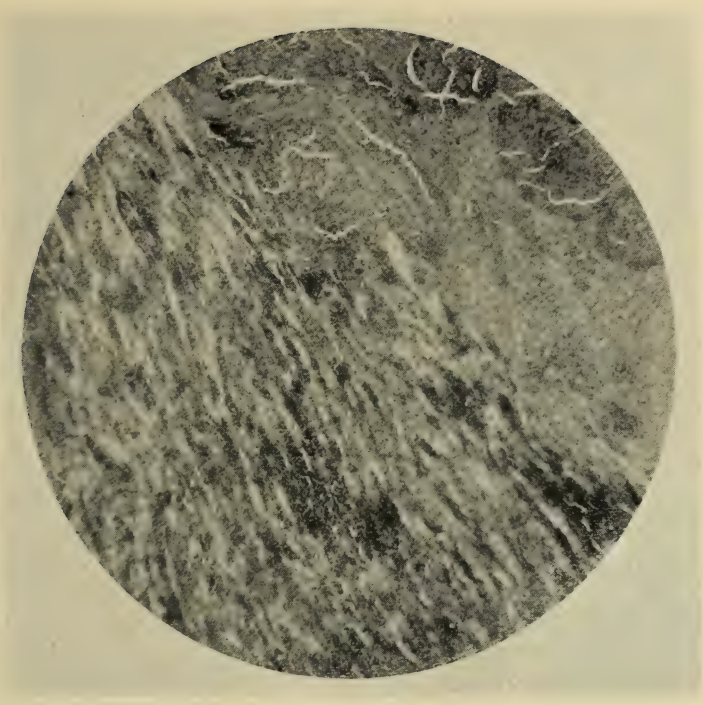

Fig. 4.—Microscopic section through line of demarcation between normal and necrotic areas of ruptured kidney. (Case III.)

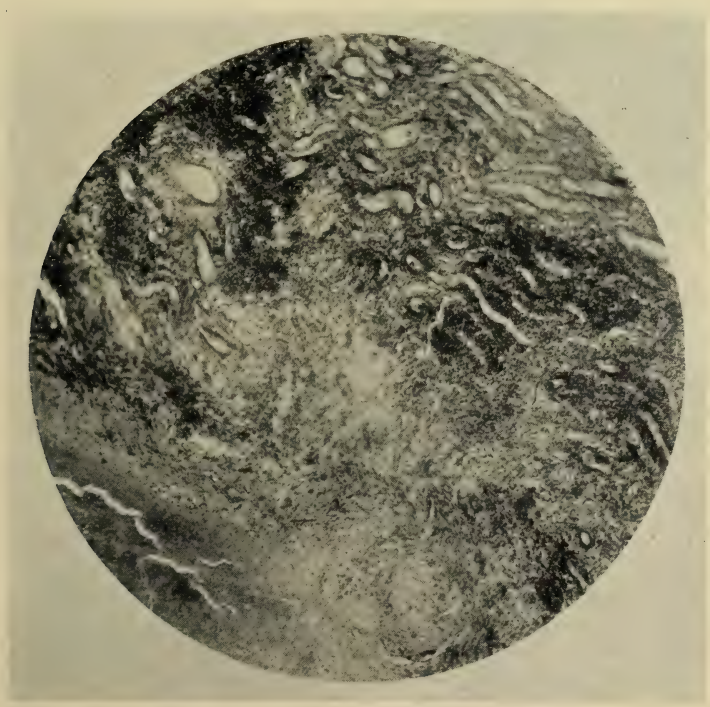

Fig. 5.—Microscopic section through line of demarcation between normal and necrotic areas of ruptured kidney. (Case III.)

which local symptoms are insignificant, constitutional symptoms are absent and slight hematuria alone directs attention to the probability of renal injury." He further claims that "of this type there are not a few, and to their almost invariable recovery can be given the largest share of credit due to the non-surgical treatment." This opinion seems to me sound and gives to expectant treatment its proper status. I have not found any pathological studies which tell us exactly what happens to the injured kidney when it bleeds for a few days and stops. Presumably it is contused or a small blood-vessel communicating with the pelvis is ruptured.

Except in those cases in which delay is demanded by reason of severe shock or complicating injuries, no just claim can be made in favor of expectant or non-surgical treatment, unless it be established that exploration of the injured kidney is inherently dangerous or more dangerous than awaiting developments. With our present kidney functional tests, our easy means of demonstrating the presence of a second functionating kidney, and our safer methods of anesthesia, the claim for such inherent or comparative danger cannot be maintained. This is particularly true when we remember that the late developments too often are anuria, hemorrhage or sepsis.

That apparent recovery under expectant treatment may be followed by remote chronic illness due to the original renal injury is borne out by three of Bugbee's cases.[1]

In Case 4 of his series a tumor of the left kidney, with dark colored, purulent urine, with very little functional activity, was found in a patient who received a kick in the side thirteen years previously, for which injury he received expectant treatment in a hospital for three weeks. In Case 6 the patient presented symptoms and findings diagnosed as organized hematoma in the lower pole of the left kidney from an injury received four years previously, since which time there had been tenderness, with attacks of dull, aching pain over the left kidney. In Case 8 more or less constant dull pain was

present in the right kidney region for eleven years following a fall from a pole, which was associated with hematuria for four days. There was present a movable right kidney with deficiency in functionating. A pyelogram showed a shadow which was presumed to be a cicatrix from healed laceration of the kidney. Kelly and Burnam mention that stone formation and tuberculosis have been observed as late sequelæ of injured kidneys treated expectantly.

Early Exploration. All operations upon injured kidneys must be in the nature of explorations, as it is impossible, without inspection, to tell the extent or character of the injury which the kidney has sustained. With early exploration, conservative treatment of the injured organ may be successfully carried out in a large number of cases in which it would probably fail if attempted later.

In a certain number of instances the lacerated or ruptured kidney may be treated by suture with entire success. This is the ideal method in suitable cases, but obviously it cannot be carried out in late operations when necrosis and infection have been added to the original injury.

Watson has collected 10 cases of suture of the ruptured kidney without fatality.[4] Connell[6] has added 3 personal cases successfully sutured, and has collected 16 cases without a death, and with only 2 failures, 1 case requiring secondary nephrectomy and 1 being followed by a urinary fistula. Connell's cases were operated on early, 1 on the morning of the second day, 1 on the afternoon of the day following the injury and 1 thirty-six hours after the receipt of the injury. A fourth case of rupture, operated on the tenth day, required nephrectomy on account of infection and gangrene.

Packing and drainage of the ruptured kidney has shown a very favorable mortality. In Watson's series, 107 cases were so treated with a mortality of 8.5 per cent. as compared with 132 nephrectomies with a mortality of 22.5 per cent.

In cases treated by packing the convalescence is prolonged, the final outcome is always in doubt, and the patient is in constant danger from secondary hemorrhage, one of Neilson's cases died from secondary hemorrhage on the thirteenth day after operation, upon removal of the last of the gauze pack. These drawbacks act as a bar to too great conservatism in dealing with an extensively lacerated kidney unless the remaining organ is distinctly incompetent.

The mortality of 22.5 per cent. for nephrectomy, as set down by Watson, in 1903, is perhaps much higher than obtains at the present time or should obtain in the future.

This mortality is dependent upon the general condition of the patient, upon the competency of the remaining kidney and upon the character of the anesthetic. Unfortunately, accidents befall the vigorous and healthy as well as those with organic disease, hence a certain mortality will always obtain.

With proper investigation of the condition of the remaining kidney, with due regard for the general condition of the patient and with the use of nitrous oxide oxygen anesthesia, few fatalities should result.

That removal of the ruptured kidney is not unduly hazardous is attested by the large number of successful cases in which the above precautions have been taken. For example, Gibson reports 4 successful cases in children;[7] Boland[8] reports 2 successful cases; my own case here reported recovered; Connell's case, already mentioned, was successful, as were also large numbers of similar cases on record.

Late Operation. It is often the case that surgical diseases and injuries do not reach the surgeon until expectant treatment has been tried and failed. In the case of the damaged kidney this means that the local and constitutional symptoms have become aggravated and that the patient reaches the operating room in worse condition than if he had been subjected to early exploration. Late operation, in the presence of a ruptured kidney, usually means nephrectomy,

though packing for control of hemorrhage and prolonged drainage for the relief of sepsis will sometimes prove successful.

Taking into consideration the high mortality and general uncertainty of expectant treatment, and the obvious limitations of conservatism in late operations, early exploration is the proper and logical method of proceeding, and its advantages may be summarized as follows:

1. The danger of exploration, in properly managed cases, is slight and is not to be compared to the risks of delay.

2. The nature and extent of the injury may be definitely and promptly determined.

3. Appropriate measures may be promptly employed for the control of hemorrhage, to guard against infection and to provide for drainage.

4. In certain favorable cases suture of the ruptured kidney may be successfully carried out, while in other instances the kidney may be saved by packing and drainage.

5. A hopelessly damaged kidney may be promptly removed, thereby shortening convalescence and restoring the patient to health in the shortest possible time.

Case Reports. Three cases of subparietal injury to the kidney have come under my observation, 2 of them in my own practice and 1 in the care of my associate, Dr. B. S. Lester, with whom I saw the case in consultation.

Two of the cases were ruptures and one I have classed as a contusion or slight laceration of the kidney, complicated by fracture of the right iliac crest, fracture of the left femoral neck and multiple fracture of ribs.

Case I.—White male, aged thirty-five years, fell from a pole, a distance of about twenty-five feet, and was brought to the hospital suffering from severe shock. Examination showed that he had sustained a fracture of the neck of the left femur, a fracture of the right iliac crest and fracture of several ribs on the right side. He was catheterized and a small amount of bloody urine was withdrawn from the bladder.

After he had reacted somewhat from shock, in order to determine the source of the bloody urine he was anesthetized with nitrous oxide and cystoscoped. The blood was found to come from the right kidney, while the left was found to be functionating normally. On account of the extent of the complicating injuries, exploration of the kidney could not be safely undertaken. For four days the urine was very scant and bloody, but after that the amount became normal and the blood cleared up and the kidney gave no further evidence of injury. No tumor developed in the loin, and I have classed the case as one of contusion or slight laceration.

Case II.—Rupture of right kidney; death without operation at end of twenty hours.

This case was seen in consultation with Dr. B. S. Lester, who has permitted me to include it with my own.

A white man, aged about thirty years, jumped from a moving train about 6 P.M., and fell, injuring his right loin. He made his way home but called the physician about nine o'clock. He was not suffering severely, but had passed some bloody urine. He was with difficulty persuaded to go to the hospital. The following morning, the blood having continued in the urine and a tumor having appeared in the right loin and his general condition having become worse, rupture of the right kidney was suspected, and he was cystoscoped for the purpose of determining the source of the blood and the condition of the other kidney. The right kidney was found to be bleeding while the left was functionating normally. The diagnosis of rupture of the right kidney was made and arrangements were made for operation. Before these could be perfected the patient became collapsed and pulseless and died before any operation could be undertaken.

Case III.—White female, aged fourteen years, was run over by an automobile on August 16, 1917. According to the history she was severely shocked and suffered from nausea and vomiting. She had great soreness in the left side of the abdomen, especially over the left kidney. Hema-

turia was present for three days, then subsided for a day and returned. She was admitted to St. Vincent's Hospital on August 21, six days after the receipt of the injury, where I first saw her.

Examination showed a temperature of 102,° pulse 112, a palpable mass in the region of the left kidney, which was extremely tender leukocyte count of 9250 and bloody urine.

The following morning she was prepared for exploration. After anesthetizing with nitrous oxide, a cystoscope was introduced and the separated urines collected. The left was bloody while that from the right was perfectly normal. Time did not permit the employment of functional tests. The diagnosis of rupture of the left kidney was made and the kidney quickly exposed.

It was found deeply lacerated and surrounded by a large clot. This completely separated the kidney from its fatty capsule. The clot was lifted out, the kidney was inspected and considered to be irreparably damaged, and was removed. One cigarette drain was used and an uneventful recovery followed. She left the hospital, with wound completely healed, on the nineteenth day.

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DISCUSSION

Dr. Stephen H. Watts, Charlottesville, Virginia.—I should like to put on record 2 cases of subcutaneous rupture of the kidney on which I have operated in the last twelve months. There was one rather striking thing about these 2 cases, namely. both of them had a high leukocytosis, although they were operated on within the first twenty-four hours after the injury occurred.

We are all familiar with the cases of extra-uterine pregnancy with extensive intra-abdominal hemorrhage in which we will find a high leukocytosis in the absence of infection, and I believe my cases were operated on before infection, at least of any extent, took place. In both of these cases the lacerations of the kidney were very extensive and the pelves were very badly torn. I think it would have been impossible for the pelves to have been sutured and restored to anything like normal, therefore I did nephrectomy in each case, which was followed by recovery without any complications.

Dr. Alexius McGlannan, Baltimore, Maryland.—Dr. McCleary, pathologist to the Mercy Hospital in Baltimore, last year described the changes that took place in a kidney the subject of a subparietal rupture. This kidney was removed from a patient who died as a result of an accident which involved, among other injuries, subparietal rupture of the kidney. He found in the area of the kidney, that apparently was undamaged, marked edema of the epithelial cells, with frequent microscopic hemorrhage—very much the condition that is described as characteristic of concussion and contusion of the brain after a closed fracture of the skull. Considering this condition, as it showed in the section in this kidney, the amount of recovery that was possible in this kidney depended entirely upon the ability of the kidney cells to take care of the pressure as a result of the edema and many hemorrhages, and it seems that destruction of the kidney, after a comparatively slight injury, is due to the edema which persists and by its mechanical pressure interferes with the circulation to a sufficient degree to cause a change in the kidney. Therefore, in injuries involving the kidney when there are signs of subparietal rupture of the kidney, early exploration is indicated, splitting the capsule to relieve

the kidney cells of this amount of pressure in the hope that the circulation will be reëstablished promptly and therefore prevent destruction of epithelium that cannot be regenerated.

Dr. Guy Le Roy Hunner, Baltimore, Maryland. — Dr. Mason's first picture made me think at once that there was a kidney half of which might possibly have been saved. It is difficult, of course, to judge what the conditions were. I should imagine, with all that hemorrhage about the pelvis of the kidney, it would be rather difficult under the circumstances to attempt to save it. But Dr. Mason has asked us to contribute our experience if we have had any with partial resection of the kidney.

I have had 3 cases of partial resection of the kidney, in one of which the *x*-rays showed a large shadow in the right kidney the size of a fist, the largest I have ever seen of a stone, and in the left kidney a smaller shadow the size of one's thumb-nail, perhaps. The functional tests in the right side showed only 4 per cent. and the other side 10 per cent. when the woman was admitted to the hospital. She had been having hemorrhage and ureteral colics from the left side. After a few days in the hospital her functional tests went up between 20 and 30 for the two sides, and I decided to operate first on the left side and remove the supposed small stone, and if she got along well we might be warranted in tackling the large stone of the right side. I was astounded when I cut in on the left side to find an immense hypernephroma, an immense tumor occupying the lower half of that kidney, and being larger than the stone shadow on right, about a double-fist-sized hypernephroma. In passing the hand around it for investigation my fingers broke into it and there was considerable bleeding, and I was rather up against it, so to speak. Of course, you could not remove the kidney in the presence of the partially destroyed right one, so I followed the expedient of splitting the dorsum of the kidney. I just split the upper half of the kidney in that fashion, V-shaped, and scooped out the tumor mass with its capsule, and then brought this lower pole of the kidney together with interrupted catgut. The only difficulty I had was with one of the vessels in taking off the lower half of the pelvis of the kidney; we took off a part of the pelvis here (indicating), and in doing so we struck a good-sized vessel. One does not tie vessels in that region before they begin to bleed for fear of robbing the kidney unnecessarily. We catch

the vessel with thumb and finger until we get the field clean, and then control the vessel by forceps until it is tied. This patient got along beautifully for a year. She was taking long automobile rides and eating as most normal people do. A year later she was shopping and slipped on the floor of the store, and in catching herself over the arm of a wooden chair she hurt her left side and had symptoms of pyonephrosis on that side. I thought it must be an accumulation along the former drainage tract, and under cocain anesthesia I opened up the old incision and found a teacupful of stone material in the pelvis. I cleaned it out and she rallied somewhat, but two weeks later succumbed from uremic symptoms.

The other cases were stones in the kidney, in which the stone collected in the lower pole and the upper pole seemed to be normal, and I did the same thing. I took the contracted fibrinous lower part of the kidney off with this V-shaped incision and saved the upper part. That was especially desirable, because these operations were done in one of the local small town hospitals in the State and I had no opportunity to investigate the cases except by plain *x*-rays. No functional tests were made and we were anxious to save as much kidney substance as possible. One of these patients had a gall-stone operation since, but both are well as regards their kidneys.

Dr. Hubert A. Royster, Raleigh, North Carolina.—I have had three cases which illustrate so clearly what the essayist has said relative to methods of treatment that I cannot refrain from mentioning them. One of the patients was a young girl who was thrown from an automobile. Her kidney was cut down upon, found torn and bleeding; it was packed and she made a splendid recovery. Another case was in a man, an alcoholic, who was also thrown from an automobile. His kidney was lacerated. I sutured it forty-eight hours after the accident, but he died from pneumonia on the ninth day after operation. A third case, the first in time of occurrence, was most interesting as illustrating the cause of the injury and the after-results. Fifteen years ago a boy was "kneed" in the left side in a football game. I saw him a week afterward in consultation. He had a mass to the left side which could be felt both on the lumbar and the abdominal aspects. His condition seemed to be good, so we let him alone for a few days and he improved gradually. He made a good recovery. At the end of five years we could not

feel anything there. Four years ago he had a severe case of typhoid fever and was laid up for three months with it. One year after that I saw him again under very distressing circumstances. He was a dissipated fellow; he had been on a night automobile ride, driving his machine and suffered an acute abdominal pain. The same doctor who had seen him fifteen years before examined him and found the mass so large that it was interfering with respiration. We opened him up immediately and found a huge pyonephrosis so big that the kidney itself was destroyed. He died a few hours after the operation. I mention this to show that expectant treatment may be very dangerous. I believe the essayist is right in saying that early exploration is the best treatment and that simple packing is probably the safest method.

Dr. F. G. Du Bose, Selma, Alabama.—I want to report one case particularly out of a small series that illustrates in itself clearly the principles of the essayist—that is, the necessity for early exploratory incision and appropriate surgical care.

A man was thrown from a wagon and the wheels of the wagon passed over him. He was carried home and his physician attended him for a period of ten days, during which time he had repeated attacks of hematuria and a palpable tumor in his left side. When he was finally brought to the hospital he had a blood count of about 1,200,000 reds, and the hemoglobin was, I think, between 25 and 30. In the day or two days while he was in the hospital under observation he would have attacks of syncope and failing pulse and collapse, and following that, or preceding it, he would void large quantities of dark, coffee-ground urine. It was not thought advisable to operate until after direct transfusion. Two brothers were secured for that purpose, and he was given direct transfusion, after which the hemoglobin was increased to 50 and his red cells to about 3,000,000. He was then operated on and the kidney removed. On removing the kidney it was found that it was extensively ruptured; it was almost torn in half, and this rupture extended well into the pelvis of the kidney. Why that man should not have died earlier of hemorrhage, particularly as some of the larger renal veins were torn in the process, I do not know. After a very stormy convalescence and after three direct transfusions of blood he survived.

Very recently I had a most interesting case of gunshot wound

of the kidney. This boy was shot in the left side and the bullet entered about an inch above the left costal margin. He was seen in a few minutes by me after he was shot. He showed signs of shock and was immediately taken to the hospital. He was given a hypodermic of morphin and atropin previously. He was fourteen years of age and an epileptic. When he arrived at the hospital his condition was remarkably good, so good that he was carried directly to the operating room and his abdomen opened on the left side of the costal arch. On opening the abdomen the anterior and posterior walls of the stomach were found perforated. The anterior opening was closed as well as the posterior opening Further examination showed a retroperitoneal hematoma. I then incised the parietal peritoneum and found the bullet had entered the kidney right at its hilus, practically preventing any possibility of resection. The kidney was drawn up through the abdominal opening and removed transperitoneally. He made a good operative recovery.

Dr. W. R. Jackson, Mobile, Alabama.—I should like to ask the doctor a question, namely, is it a fact that cortical contusion of the kidney invariably results in hematuria? I do not think this happens always. We can have the cortex of the kidney contused almost to the point of complete, we might say death of the tissue without any hematuria.

In regard to blood-clot extension I can think of two causes: blood-clot in the tubuli uriniferi, plugging them up and stopping the secretion of urine from behind and remaining *in situ* permanently and necrosis with blood and no hematuria. Ordinarily, however, the more extensive condition due to shock, without rapid vibration of the cells of the kidney substance itself, results in hematuria. Very often, therefore, we have hematuria from concussion of the kidney, and when you operate you find nothing particularly the matter with the kidney. When we have marked hematuria and shock we explore the kidney, but in so doing we may find nothing. I have seen that two or three times. I have explored the kidney and found it engorged and congested, and that is all.

Dr. Mason (closing).—I wish to thank the gentlemen for so freely discussing my paper. I was particularly anxious to present a paper on this subject, because in looking over the index to the *Transactions* I found that it had not been discussed

by the Association, and I felt sure that we would have a number of case reports and a free expression of opinion. I knew Dr. Hunner had had experience in resections of the kidneys. I was not satisfied as to how far one could go with resection, and so I wanted the views of the members of the Association regarding this point. I am very glad to have heard these reports.

The pathological report mentioned by Dr. McGlannan has some bearing on what I have said, and he answered pretty directly the question asked by Dr. Jackson. In a small cortical contusion you will not necessarily have hemorrhage; you would have pain and some other symptoms. In a case of that sort the proper procedure would be to split the capsule of the kidney and drain as Dr. McGlannan has suggested. Even in an injury that causes considerable contusion of the kidney you have to deal with a microscopic hemorrhage whether you have an open hemorrhage or not.

The cases reported by Dr. Royster bring out the point I mentioned in regard to apparent recovery from these injuries without operation, but it is likely that if they are followed up it will be found that some apparent recoveries are followed by sequelæ which produce a great deal of invalidism, finally leading to fatalities.

I think Dr. Du Bose's case proves the point that I contended for, namely, that these cases should be taken out of the hands of the general practitioner and put into the hands of the surgeon at once; because they all are surgical cases and I do not believe many surgeons at the present time would treat them expectantly for very long.

SOME RECENT LITERATURE ON SHOCK

By F. W. Parham, M.D.
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In an address on "Shock and its Management," in 1913,[1] I said: Since Crile opened up the new era in shock investigation in 1889 we have acquired a vast deal of information, but who can say we have arrived at ultimate truth? At that time Crile's vasomotor theory seemed the most reasonable and the most practical explanation of shock, and even today, though the foundation has been seriously undermined, the therapeutic deductions consistently drawn from his investigations remain the most satisfactory that have so far been suggested.

We are living in the most stirring period of the world's history, and while science on the one hand has been busy in evolving the most destructive engines of war, it has, on the other hand, shown itself most wonderful in the development of the agencies intended to preserve life and ameliorate human suffering. The medical profession in this war has made itself felt as never before in the history of the world. Dr. Crile recently characterized the war machine thus: "One part of the machine blasts and breaks and tears, terrorizes and kills; the other part conserves and discovers, assuages and relieves, reclaims and rebuilds. This is the medical part, our part, of this war machine!"

Many new problems have arisen and old problems are finding new solutions. Antisepsis has been again revived,

for new conditions confront the surgeon, and his ingenuity is working out new plans of treatment because the old plans have failed. The saving of life and the mitigation of suffering are the watchwords now. The problems of shock are again in the forefront and surgeons and laboratory workers are busy as never before, for the needs of the wounded are greater than ever before. Coöperation is the order of the day in every phase of defence.

At the request of the Committee on Physiology of the National Research Council a coöperative investigation of surgical shock has been instituted in a number of physiological laboratories throughout the land and many independent investigations are being carried on. Under the impetus of the necessities of war this work is proceeding steadily, and already the fruits are apparent. Since this world war began many valuable papers have been published and the literature of the past three years has illuminated many of the obscure phases of shock, if it has not settled them. Cannon,[8] in his Shattuck Lecture, has discussed, in a most interesting and instructive way, the signs and symptoms of shock and has attempted a classification and analysis of its phenomena. He examines them under four heads: The disturbances of sensation, of motion, of respiration and of circulation. He rejects Meltzer's theory of central inhibition as untenable and unnecessary. In a state of shock the blood-pressure is low, and evidence is at hand that increased synaptic resistance is present—that is, there is a blocking of the afferent pathways to the central cells, requiring increasing stimulation to pass the raised synaptic threshold. Inadequate blood supply impairs the nutrition of the muscles and the nerves innervating them, thus explaining the general relaxation, the irregular and feeble movements and the slowing and weakening of central nervous function. Fall of temperature, thirst, sweating and pupillary dilatation are to be regarded as secondary.

The superficial rapid character of respiration at once refutes the idea of Yandell Henderson that undue pulmonary

ventilation caused by pain is the primary factor in shock. Henderson himself seems now to contend less strenuously for his theory.[20] Even acidosis, which may, by irritation of the respiratory center, produce increased frequency of respiration, must be considered secondary to some other cause. The circulatory signs of shock are a low arterial pressure, pallor, small thready pulse and a cold skin. To explain these the cardiac factor, the vasomotor factor and the factor of blood volume must be studied. The cardio-inhibitory paralysis theory of Howell is disproved by the reflex slowing of the heart following stimulation of the central end of the cut vagus and by the dropped beats caused by adrenalin. That the heart is not at fault is shown by its acting as soon as supplied with blood. Low arterial pressure may affect the heart and increased hydrogen-ion concentration of the blood causes cardiac relaxation. "The important considerations are to increase the volume of well-oxygenated blood so as to lessen the dangers of acidosis and to raise arterial pressure to such a degree as to provide a proper flow through the coronary vessels."

The cardiac factor is therefore not primary in shock nor is the vasomotor factor. The experiments of W. T. Porter, of Seelig and Lyon, of Seelig and Joseph, of Frank Mann, of Morrison and Hooker, of Yandell Henderson and many others show this center to be not paralyzed but in a state of *increased* activity, and Pike, Guthrie and Stewart have shown it more capable than any other center of withstanding the influences of anemia. Low pressure does not prove its exhaustion, for this follows hemorrhage also. Cannon concludes that "we must respect the vasomotor center as an agent whose capacities for continued action are its most outstanding feature."

The real explanation for the circulatory disturbances is to be found in diminished volume of blood in circulation. The same thing occurs in hemorrhage, but the explanation is different. In hemorrhage the blood is actually lost from the

body, while in shock it is in the body but not in circulation. There seems to be general agreement that it is locked up in the portal system. Clamping the portal vein causes the same fall in pressure as in severe bleeding, and Mann and Morrison and Hooker have pointed out the distention of the mesenteric veins in shocked animals.

Frank Mann[9] has published some experiments to show that a large amount of blood is retained in the splanchnic area in shock. He set the problem to determine the actual loss to the circulation. He ascertained first the total amount of blood to body weight. This he estimated at 7.7 per cent., or one and one-thirteenth of body weight. He thus established the normal standard. He then bled the animal from a large vessel; this represented the measure of efficient blood. He then drew the blood from the venous side (the right auricle), which, added to the arterial blood, made up the mobile blood. The sum of these two subtracted from the calculated total blood represented the immobile or useless blood. In unshocked animals 24 per cent. could not be drawn and was considered immobile blood. He found that in shock produced by opening the abdomen and manipulating the intestines, 61 per cent. remained immobile. Compare this with complete destruction of the vasomotor center, which immobilizes only 42 per cent. In shock the amount of circulating blood is less than in section of the spinal cord. A series of experiments confirmed these statements. This immobilization of blood takes place beyond vasomotor control.

A memorandum upon shock and some allied conditions issued by the Medical Research Committee,[7] February 27, 1917, furnishes some exceedingly valuable data intended to aid in the elucidation of this complex problem.

Dale and Laidlaw's experiments with histamin are fascinating in their interest. They find by the use of the hemoglobinometer and the hematocrit "that the profound fall of blood-pressure produced by this substance is accompanied by

a striking concentration of the blood, amounting in some cases to a loss of one-half the original volume of plasma in about five minutes." But this is not the only way in which the blood volume in circulation is reduced.

While the evidence seems to point to constriction of the arterioles in shock, "they provisionally attribute the shock in the first instance to a widening of the whole capillary area in the viscera and the musculature." They instance Colton and Slade and Lewis (Heart, 1917) as furnishing evidence of "active contractility of the capillaries." Dale and Laidlaw suppose that, under the action of the poison, the capillary tone is lost, "so that the blood from the arteries being diffused and stagnant among the slack capillary channels the quantity of blood reaching the veins is inadequate for the filling of the heart."

About the main fact that there is depletion of the macroscopic vessels, both arterial and venous, there can be, they say, no room for doubt; it will be obvious how this deficiency will be aggravated by the direct reduction of blood volume by loss of plasma to the tissues.

The loss of plasma, too, increases viscosity, which still further impedes the flow. This impeded flow leads to defective oxidation, with resulting acidity, and so to further loss of water by osmosis, as suggested by Henderson. Frank Mann likens the phenomenon to the stasis of inflammation.

Lastly, the fall of arterial pressure, due to defective filling of the heart chambers and the increased viscosity of the blood, will seriously interfere with the coronary circulation and, consequently, the nutrition and oxygenization of the heart muscle itself. A series of "vicious circles" is set up.

Dale and Laidlow, impressed by the similarity of these phenomena to those of shock, made the experiments with animals in shock and found similar phenomena developing.

Captain Marshall, R. A. M. C., made some observations in France on the hemoglobin content of blood, which seem to support the view that oligemia is an important factor.

Experiments are also reported of Dale and McIntosh with organisms of the gas-gangrene group isolated from human material.

The evidence available indicates a striking similarity of origin underlying the similarity of symptoms between the toxemia of gas gangrene and other shock-like conditions.

Effects of the same kind are produced by other products of protein digestion, bacterial products, etc.

Bainbridge and Trevan also are referred to as conducting experiments on the effects produced on the systemic and portal circulations by repeated injections or by long-continued, slow infusion of adrenalin. The same factor of concentration of blood has presented itself. The inquiry was suggested by the observation of Elliot, Cannon and others that extreme emotion or severe pain, factors in some cases of shock, are accompanied by an outpouring of adrenalin. The significant fact was made out of a great rise in portal pressure, the systemic venous pressure remaining unaltered. This was accompanied by great loss of plasma and increased viscosity, as shown by the relative increase of the corpuscular element. There was evident obstruction in the liver. The nature of the obstruction will be the subject of further investigation.

Mann[9] contends that shock and hemorrhage are identical in all essential particulars. In some respects, however, most important for the purposes of the surgeon, the two conditions differ materially, as has been clearly pointed out by the British Research Committee. In hemorrhage, even of sufficient severity to diminish the output of the heart and lower the blood-pressure, if the case be not allowed to go too far, normal reaction may yet take place, fluid be drawn back into the vessels and restoration be established, and this we know may be enormously assisted by transfusion of blood or by saline infusion. In shock oligemia is pronounced, the outgo of plasma into the tissues continuing in spite of the introduction of fluids into the blood, and only the most temporary

benefit is accomplished. So long as a man is alive there is hope in hemorrhage if fluids can be introduced. So bad, however, is the picture in shock that Rendle Short declared in 1914 that it was hopeless to do anything for the shocked victim. In hemorrhage, too, cardiac stimulants have a place, but in shock they are worse than useless until reaction can be otherwise established. Another important difference is the effect of local anesthesia in preventing shock by blocking the centripetal impulses.

The various experimental results in this report "suggest that the removal of blood from effective circulation may be brought about in more than one way" by blood poisons, such as histamin, protein substances, bacteria or their products, such as the gas bacillus of Welch, as well as by hemorrhage and traumatisms. We might add to these caisson disease, in which air embolism occurs (Pike[15]), and Simonds[16] calls attention to two very widely used methods of inducing a condition of low blood-pressure simulating surgical shock in man—namely, peptone poisoning and fat embolism. In both these the fall in blood-pressure is marked and the condition cannot be readily distinguished from shock. There are some differences which seem to favor the statement of the British Research Committee that there may be different forms of shock and that the forms are related to the cause. In peptone shock the blood accumulates in the liver and the right heart is empty. In fat embolism there is general venous stasis and both right auricle and ventricle are dilated, owing to the pulmonary obstruction. There is loss of tonus in the one case in the pulmonary and in the other in the splanchnic vessels.

May there not be a special traumatic type of shock marked by obstruction in the liver, as shown by swelling of this organ and congestion of the portal system? The work of Simonds seems to indicate that this may be differentiated from another form of shock without this feature. May not traumatisms of different parts of the body affect differently various areas

of the vast capillary system if the views of Colton, Slade and Lewis that capillaries have contractile power of their own are to be accepted, so that in the one case we may have primarily the portal circulation, in another the pulmonary and in another the intracranial area affected? As surgeons we must, with Bloodgood,[12] take issue with Mann,[10] who asserts that shock can only be induced in an animal under ether by opening the abdominal cavity and irritating the intestines. If it be correct that in an animal under ether no amount of peripheral irritation will bring it into a state of shock, I am sure most active hospital surgeons have seen many a man reduced to dangerous, often fatal, shock by severe crushing of limbs or head. What is the experience of this war? W. T. Porter[3] remarks that in beginning his study of traumatic shock in France and Belgium he asked what wounds were most often followed by shock, and was told by the surgeons of Compiègne and La Panne that it was most frequent after shell fractures of the femur, less so after injuries of the smaller humerus and small bones. Again, in his third article, published September 6, he reports that of numerous wounded "the only shock was that caused by abdominal injuries, fractures of the femur, or by multiple wounds of the subcutaneous tissue." He substantiates these statements in his fourth communication[5] (September 6, 1917). Archibald McLean,[19] who spent eighteen months in service in France, reports in a paper read before the American Surgical Association: Practically all the wounds "were of the locomotor system or of the abdomen." Porter, in his fifth communication,[6] states that "in over 1000 consecutive cases of shock no instance was encountered except in severe abdominal injuries, fractures of the femur and multiple severe wounds of the subcutaneous tissues. "In the abdominal cases he thought the mechanism was clearly direct injury of the great splanchnic system. In the other two types the cause was in his opinion fat embolism. The shock in the fat embolism cases came on later, several hours

or even days. Bissell,[17] referring to Bloodgood's criticism of Mann's statement that he cannot agree that abdominal exposure is the only way to cause shock, argues that the cases spoken of by Bloodgood were perhaps cases of fat embolism. He calls attention to the masterly descriptions of Park, thirty-three years ago, and expresses the opinion that Bloodgood's cases and those of Park might be interchangeable so closely do they resemble one another. So that, as he seems to suggest, the crushed limbs and wounds of the soft tissues generally should be explained by fat embolism and the abdominal cases as pure shock. This is the conclusion to which Porter[3] comes after studying the various kinds of wounds seen in France. Pike[15] writes: "Whatever the other conditions in traumatic shock may be we find strong evidence for the view that some physical damage within the central nervous system must be included among them. No return of function, or only an extremely slow and imperfect return, is possible, so long as the blood-pressure remains low. Neither low blood-pressure of the magnitude recorded in cases of surgical or traumatic shock nor the diminished supply of blood to the brain is sufficient in itself to produce such speedy death as is often reported."

Guthrie,[21] as a result of his very thorough investigation of all the phenomena of shock, feels inclined to attribute the earlier stages "largely to degradation and fatigue of the bulbar centers," which seemed to show greater anemia than that of the higher centers, as indicated by the persistence of eye reflex. The evidence seems to indicate "fatigue of the nervous tissues as a causative factor of considerable magnitude."

The treatment of shock resolves itself into measures intended to raise the blood-pressure and restore the animal heat. W. T. Porter[2] found remarkable absence of nervous shock in the midst of the most horrible carnage, so that the psychic factor seemed to play a very small role. In the badly wounded he found the blood-pressure a most valuable guide, especially the diastolic. When the diastolic pressure fell below

50 mm. the condition was very grave and resuscitation was doubtful, but when reaction could be brought about recovery was often rapid. Elevation of the lower limbs and head lowered just below the abdomen was the best position. A special table was devised, which enabled him to keep the patient warm in the proper position. Saline infusion was given for temporary effect and sometimes adrenalin given in weak solution continuously was advantageous, but the saline infusion was temporary in its effect unless the blood-pressure could be raised. Porter[4] speaks highly of respiratory suction or the thoracic pump. By introducing carbon dioxide into the air breathed the chest was expanded and a powerful traction force exerted on the blood in the liver, drawing it through and establishing reaction in some very bad cases. Cannon,[8] recognizing that the retention of the blood in the splanchnic area was due to the obstruction in the liver, tried pituitrin diffused over the portal vein, with the intention of producing contraction of the smooth muscle and forcing the blood through. Adrenalin, in his opinion, was contra-indicated because it contracted the portal terminal vessels in the liver. It is doubtful if the action of pituitrin can be limited to the portal vessels in the abdomen. Cannon's recommendation would necessitate a certain amount of manipulation in order to deposit the pituitrin, and in any but an abdominal operative case it would require a special incision. For this reason, then, and also because it would be impossible to confine its action to the portal radicles, it would seem an impracticable procedure. The suggestion of Porter is simple and worthy of trial, because so easy to apply.

Finally, as the alkali reserve is probably reduced in all these cases, the administration of bicarbonate of soda might be tried with advantage. Dawbarn's suggestion of bandaging the limbs employed in conjunction with the respiratory suction is valuable. This would have the effect of drawing the blood to the right heart not only from the vena cava but also from the portal capillaries. There is one danger sug-

gested by Simonds: The suction might in fat emolism draw the fatty globules from the lung to the left auricle and be there sent with fatal effect to the brain.

In conclusion, shock must be considered a disordered condition of the higher nerve centers, due to the centripetally carried impulses of irritation resulting from traumatism; the lowered blood-pressure, diminished sensibility, pallor, cold skin and feeble pulse are to be explained by the diminished volume of blood sent out by the heart, owing to its being locked up principally in the splanchnic area. In operative work it may be prevented by local analgesia whenever it can be applied, and in the traumatisms of accident or war is to be treated by quieting by morphin the irritation of the nervous centers, by favoring the flow of blood from the stagnant splanchnic area to the heart and brain and by the applications of heat to the surface. The flow of blood to the heart is favored by elevating the lower part of the body and by utilizing the thoracic pump by the stimulating effect on respiration of carbon dioxide, as suggested by Yandell Henderson and Porter.[4]

Stimulants seem to have no place in the treatment of shock, but the use of saline infusion or the transfusion of blood with the aid of such agents as pituitrin to raise the blood-pressure may be of temporary benefit. Hypertonic salt solution, with the addition of the calcium salts, will be better retained in the vessels than normal salt solution. Hogan's gelatin solutions or Bayliss's addition of acacia to the fluid seem to offer some advantages over the simple physiological salt solution; but since the escape from the vessels of the blood plasma is a prominent feature in pronounced shock, as shown by the British Research Committee, it can hardly be expected that any of these solutions, even blood itself by transfusion, will remain in the vessels under aggravated conditions of shock unless reaction can first be brought about by the measures suggested by Porter.[2] Such a condition of shock is practically hopeless.

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DISCUSSION

Dr. J. Shelton Horsley, Richmond, Virginia.—This is such an excellent up-to-the minute review of shock that it is very instructive. There are one or two points I would like to speak about: first, one of the last recommendations that Dr. Parham makes, the Hogan solution. Three years ago, at the meeting of this Association, I reported cases in which I used the Hogan solution of colloid. That it restores blood-pressure very promptly is undoubtedly true in every case, and the immediate result is really quite wonderful, almost as dramatic as in transfusion; but three of my four cases died of kidney symptoms. There was a prompt recovery in every case temporarily. While the colloid will stay in the circulation and prevent diffusion, it undoubtedly has some effect on the terminal capillaries of the kidneys which, sooner or later, is deleterious, and in many cases probably fatal.

A few weeks ago, at the Mayo Clinic, I heard Dr. Mann read a letter from Dr. Cannon, who is at the Front, in which he stated, if I understood the letter correctly, that in addition to capillary stasis in shock there was some acid condition accompanying it, and that the administration of bicarbonate of soda, even in small doses by mouth, seemed to produce wonderful results. The work of Bissell in showing that a great many cases of pulmonary fat embolism produced symptoms identical with the symptoms of shock is interesting in that connection.

Dr. William C. MacCarty, Rochester, Minnesota. — In regard to the use of bicarbonate of soda in cases of fat embolism, for instance, I do not believe that bicarbonate of soda will have any real effect upon the fat in the blood. The injury has already been accomplished when the condition is diagnosed, and I believe any attempt to produce a beneficial effect by bicarbonate of soda is almost too late. However, if it has any effect at all it might be utilized as a preventive measure.

I have been interested in cases of fat embolism connected with shock, and especially the work of Bissell, who was formerly a pupil of Dr. LeConte and is now in charge of autopsy work. Some of the cases which we surgically speak of as shock, whether operative or traumatic shock, are due to minute quantities of fat which have gotten into the circulation. Bicarbonate of soda might do some good as a preventive measure, but after the vessels are filled with fat the damage has been done. That is my judgment from what I know of Dr. Bissell's work in connection with fat embolism. Further than that my opinion is not worth much.

Dr. William M. Jordan, Birmingham, Alabama.—Like the rest of the members of the Association, I have appreciated and enjoyed Dr. Parham's presentation of this important subject. I think we all agree that in the present emergency there can be no more important topic brought before us.

It was my good fortune this fall to see some experimental work in the treatment of shock under the direction of Dr. Edward Martin, showing two points, the value of blood-transfusion, and the utter futility of drug stimulation. We tried out the usual line of stimulants with the view of noting variations in blood-pressure, but found no effect whatever from such stimulants as strychnin.

We secured a very temporary effect from saline infusion, and noticed an enormous but very evanescent rise when adrenalin was added, but the most benefit we got from any drug treatment you might say was from soda solution given intravenously. That, however, was not maintained long enough to be regarded as satisfactory, and we came to the conclusion, after observing the sustained benefit of blood-transfusion, that this must be the sheet-anchor in the treatment of profound shock, in the light of our present knowledge.

Dr. Crile gave a most interesting clinic at the University

Hospital bearing on this subject. In the first place, he showed the harm that might come from the too rapid infusion of salt solution, and actually killed a dog before the audience, in a few minutes, by rapidly introducing salt solution under pressure into the vein, and proceeded to explain how death was brought about and pointed out the danger of the unwise use of saline infusion. Water introduced into the vein under pressure goes through the circulation and is secreted into the intestinal canal with great rapidity. Dr. Crile opened the dog's abdomen and demonstrated the enormous quantity of water that had accumulated in the bowel. In a second demonstration, after blood transfusion, he was able to show a high rise in pressure, and the more important fact that it was maintained, and to our amazement it was still maintained after the medulla was destroyed and the animal was kept alive by artificial respiration.

Just a word more in reference to one interesting point brought out by Dr. Parham, namely, as to the condition of the circulation in the liver in cases of shock. I have been thinking on that line for some years in view of the fact that, it seems to me, we get a better effect from the proctoclysis method of administering saline than we have obtained by the intravenous plan. In seeking for the cause of that the only difference between the two is the fact that in the proctoclysis method the saline went through the liver rather than into the circulation directly. I have never made any experiments along that line, but in one abdominal case which seemed to require venous infusion I used saline infusion through a mesenteric vein. I asked Dr. Plant, who was conducting these experiments, to consider the method with a view to making comparative observations on the relative value of infusion in the general venous system as compared with that of the mesenteric vein. In that case the infusion might be suppossed to dilute the accumulation in the liver and possibly give us a more lasting benefit. I think in these cases when the abdomen is opened that idea might be tried out.

Dr. F. G. Du Bose, Selma, Alabama.—I want to commend the use of bicarbonate of soda. Clinically it has served me a better purpose than any one measure, but in very dilute solution. In the hospital I use a concentration of 10 per cent., taking a 100 c.c. of solution and introducing it into the vein directly. The experiments carried out and published two years ago in the *American Journal of the Medical Sciences* showed that concen-

trated solutions have the same effect on the heart muscle as adrenalin, which carries the blood-pressure quickly to a high point, and almost as quickly it drops below the original blood-pressure; whereas bicarbonate of soda differs in effect that it carries it as high as adrenalin and gradually it declines. With a large Luer syringe, so soon as the blood-pressure declines to a point near its original measure in shock, a second hypodermic of bicarbonate of soda is given. The injection of large quantities of fluid into the veins for shock has not been as satisfactory to us as the smaller and more concentrated solutions.

Another thing that has not been mentioned is that morphin given in small doses, say one-eighth of a grain, is the best heart stimulant we have.

Dr. J. Shelton Horsley, Richmond, Virginia.—It was suggested that the soda might saponify the fat. I would like to add one word as to the advantage of Locke's solution over the ordinary salt solution. It is one-tenth of 1 per cent. glucose, in addition to sodium and potassium salts; is retained in the vascular system longer than salt solution, and has an element of nutrition at the same time.

Dr. Parham (closing).—I have no knowledge of Dr. Cannon's latest communication in regard to the use of bicarbonate of soda and its effect on fat embolism. It seems to me that its use is simply for the restoring of the alkaline reserve which is so much reduced. When the alkaline reserve is so much reduced, something should be done to restore that in order to accomplish any results. I do not see how it can have any effect upon the fat circulating in the blood.

We are still in a state of great confusion in regard to the causation and management of shock. One gentleman suggested by preference the use of saline infusion by rectum. If Cannon is correct that there is obstruction in the liver, that is the worst way to give it, yet we see good effects following the solution.

Following Dr. Trout's suggestion made some years ago I rarely ever use salt solution by the Murphy drip, preferring to use tap water with sugar, and Dr. Horsley's suggestion there is much to the point. In some cases of shock, for the purpose of restoring the function of the kidney, I have used 33 per cent. of sugar and have seen no ill effects from it. It has a

diuretic effect and following some observations I have seen in the recent literature, the use of sugar intravenously may show some advantage over salt solution. In the use of salt solution I believe the latest suggestion is that a hypertonic solution should be used intravenously, and not the physiological solution, because we want to favor osmosis into the blood and some of the recent discussions seem to indicate that hypertonic salt solutions (5 to 10 per cent.) might help to retain the fluid in the blood.

In regard to the suggestion that Dr. Horsley has made, that the Hogan solution may give rise to subsequent trouble, it seems to me that is quite reasonable. I have not used the Hogan solutions, so that I have had no experience with it. It strikes me that if these suggestions in regard to the use of hypertonic salt solution are correct it would be proper to confine ourselves to that. Unfortunately, there is no fluid in a certain stage of shock that will be retained in the blood. The oligemia is so pronounced that the serum and plasma go rapidly out into the tissues due to the condition of acidosis which is induced by the depletion of the alkaline reserve in the blood.

I was much impressed by the observations which Porter made in reference to shock near the battlefield. His latest communication is in regard to the use of the thoracic pump by causing retention of carbon dioxide. The respiratory apparatus then acts as a suction pump, drawing the blood into the chest and through the liver, thus emptying the splanchnic system. He reports that he has restored some people from a condition of almost hopeless shock. He gives a few cases in detail.

Some years ago Yandell Henderson, who was a member of the Committee on Anesthesia of the American Medical Association, suggested in our operative work it might be a desirable thing in certain cases to introduce a certain percentage of carbon dioxide into the anesthetic we were using, about 5 per cent. (Porter says to increase it to about 3 per cent.), so as to start the action of this thoracic pump. Yandell Henderson a few years ago held the theory of increased pulmonary ventilation, which he said was due to the action of pain in increasing pulmonary ventilation and causing a depletion of CO_2; but he was clearly wrong in this, as it is well known that in shock, respiration is more superficial than normal.

THE USE OF BULL'S SERUM IN THE TREATMENT OF WOUNDS INFECTED WITH THE BACILLUS AËROGENES CAPSULATUS WELCHII

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In 1892 Welch and Nuttall[1] described an anaërobic encapsulated bacillus isolated from a body, which at post-mortem examination showed the presence of emphysematous crackling in various situations. For several reasons it was argued that the emphysema was the result of an antemortem infection and not a cadaveric decomposition. Animal experiments with the organism proved this argument to be based on fact. The organism was named by its discoverer the Bacillus aërogenes capsulatus, since shortened by popular usage to Welch bacillus.

Later studies were made by Welch and Flexner[2] and the clinical application made by Bloodgood,[3] who pointed out the principles involved in the correct treatment. Several other clinical reports found their way into the literature, but the subject did not attract great attention until the present war brought out an immense number of cases of infection with the organism, 70 per cent. being reported by Taylor[4] and nearly 90 per cent. by Fleming.[5] From such frequent infections the number of cases of gas gangrene has been very large.

Considerable success may be obtained in preventing gangrene if the infected wound is seen and treated early.

However the rapidity with which the infection and gangrene may develop is shown in Bowlby's report[6] of a case in which the gas was present five hours after the wound was received, gangrene and death following in sixteen hours.

The organism grows on dead muscle and similarly damaged soft tissue, and the infection spreads along the sheath of the large vessels and nerves. The treatment of the infection therefore requires the removal of all such damaged tissue, the opening up of the course of the vessels and the use of suitable disinfectants. Of the antiseptics employed, Dakin's solution is the most efficacious for reasons to be given later. The surgical removal of the damaged tissue will often require the nicest judgment in deciding between excision and amputation.

The infected muscle is easily recognized by its loss of contractility and its dirty brick red color as distinguished from the purple brown of the normal tissue. The usefulness of the limb after removal of all such tissue becomes an important factor in deciding for or against amputation.

The diagnosis of the infection is not difficult. The organism may be recognized in cover-slip preparation and in twenty-four-hour cultures. Routine examination of all suspicious wounds by these methods will permit a diagnosis in many cases before the development of gas and of gangrene.

Several theories have been advanced for the development of the gangrene and of the toxemia in this infection. The decomposition products of the infected tissue—endotoxin absorption, acidemia and antitryptic action—have each been advanced as the cause of the destructive action and fatal termination of the infection. Kenneth Taylor[7] advanced a theory that gangrene was produced by the pressure of the confined gas on the bloodvessels, causing necrosis, the necrotic tissue then being disorganized as the result of invasion by putrefactive organisms. He argued that the Welch bacillus acting as a saprophyte attacks the carbohydrates of the muscle producing the great quantity of gas required for the

pressure. Taylor makes a contrast between the action of the tetanus bacillus and that of the Welch bacillus in the body.

Carrol G. Bull and Ida Pritchett,[8] of the Rockefeller Institute, not only describe the true action of the Welch bacillus in producing gangrene and toxemia, but also publish the production of an antitoxic serum.

This study proves that a highly potent soluble toxic agent is regularly produced by the bacilli on which their poisonous or lethal action depends. This toxic agent is an exotoxin, and in this manner resembles the toxin of the bacillus of tetanus. The toxin produces two sets of effects, according to the manner of injection into animals.

(*a*) Hemolysis following intravenous injection.

(*b*) Inflammation and necrosis of subcutaneous tissue and muscle follows injection into these structures. In this action the toxin resembles the effects produced by the bacteria themselves. Suitable injection of a rabbit with the filtrate of a culture of the bacillus produced a blood serum capable of neutralizing the toxic substance causing the inflammation and necrosis and also neutralizing the specific hemolysin. It is interesting to note that *in vitro* five minutes' contact with Dakin's solution will destroy the toxin.

In an infection with the Welch bacillus it is shown experimentally that the injection of this antitoxic serum will prevent the development of the spores into the vegetative bacilli, and also that the serum will deprive the vegetative bacilli of their toxic products, which now appear to be their real offensive instrument.

The chief danger from infection with the Welch bacillus is essentially similar to that following tetanus infection. In both cases the toxic agent is an exotoxin yielded by the multiplying organisms at the focus of infection. There is, however, great difference in the local action of the two toxins. Tetanus toxin has little local effect while the Welch toxin produces marked inflammatory and necrotic change

in the local tissue. The Welch bacilli therefore grow abundantly, producing wide destruction of tissue, in which process they are soon assisted by the ordinary pyogenic micro-organisms.

We have therefore in the presence of gas gangrene two processes to combat:

1. The local growth of the bacteria which produces the toxin and is in turn assisted by the action of this toxin on the tissues.

2. The systemic effect of absorption of the toxin. The extent of this absorption is easiest measured by the degree of hemolysis shown by the diminution in the number of the red blood cells. The temperature and pulse-rate also indicate the extent of toxemia.

Injection of the antitoxic serum will neutralize both processes. The progress of detoxication may be followed by counting the increasing number of red corpuscles. When sufficient antitoxin has been administered the increase will be rapid; a difference of from 500,000 to 1,000,000 cells will be noted in two hours. With this increase there will be a fall in the temperature and pulse-rate.

As soon as the condition of the patient warrants operation the gangrenous tissue must be removed by amputation or excision. This is important, because reintoxication has seemed to occur by absorption from the necrotic tissue. When amputation is done the operation must be a circular amputation without flaps: the guillotine method.

The dose of antitoxin used is from 5000 to 15,000 units, 7500, as an average, given intravenously. At the same time about the same quantity is divided between 4 or more intramuscular injections given proximal to the area of infection. The intravenous injection should be repeated in two hours if no improvement is shown in the temperature, pulse-rate and more especially by the increase of red blood cells. Anaphylaxis is not common when the successive injections are given at short intervals of time. The development of an

anaphylactic shock under such circumstances, in the presence of gas gangrene, seems to indicate a fatal infection.

Through the kindness of Dr. Flexner and of Dr. Bull I have had the opportunity of treating 3 cases of gas gangrene with the serum. Three cases are reported in abstract now.

Case I.—Mercy Hospital, No. 3364. The patient was a white male, aged fifty-two years, who was admitted to the hospital September 20, 1917, following a wound of the right buttock and thigh inflicted by a shotgun at short range thirty-six hours before admission. His temperature was 103°, pulse 120, expression anxious and general appearance that of a patient suffering from a septic infection. The wound of entrance, situated just behind the great trochanter, was small and surrounded by a dark brown area of necrotic skin, about 3 x 5 inches, which area crackled on pressure. The exudate on the surface of the wound contained bubbles. There was brawny induration extending back to the tuberosity of the ischium and down the thigh for a distance of nine or ten inches.

Immediate operation was performed. An incision was made extending above and below beyond the indurated area. The necrotic skin was excised. The felt wad of the shell was removed, and with it many small shot. The gangrenous tissue was excised until the floor of the wound showed only clean muscle and fascia. A number of bloodvessels were tied with chromic catgut. Carrel tubes were introduced, the wound dressed and instillations of chloramin made every two hours. The wound was dressed daily and looked healthy, smears showing a few bacilli for the first few days. In spite of this condition of the wound the patient remained toxic, and therefore on September 22 he was given 30 c.c. of antitoxin into a vein of the arm. A similar dose was given on the next day. The patient's condition continued uneventful until the fourteenth day, when he developed lockjaw. Five thousand units of tetanus antitoxin were given into his spinal canal and the symptoms disappeared within the next twelve

hours. On the eighteenth day it was noted that the fascia lata appeared necrotic. Smears taken from this area showed gas bacilli. Another dose of the antitoxin against Welch bacillus was given on the twenty-second day and was followed by an anaphylactic reaction. On the thirtieth day the wound was partially closed by suture and the patient then made an uninterrupted recovery.

Case II.—Mercy Hospital, No. 4242. The patient was a white man, aged thirty-nine years, who was admitted November 7, 1917, on account of a compound comminuted fracture of the right leg and closed fracture of the left leg, caused by an automobile accident. The wounds were disinfected with iodin, the fractures immobilized in padded splints and the patient was given a prophylactic injection of tetanus antitoxin. At 8 P.M. on November 8 it was noted that the patient's temperature had risen suddenly to 102°. The resident surgeon, Dr. Kearney, at once examined the wound of the right leg, and noting an emphysema and discoloration, made a cover-slip preparation which showed the Welch bacilli. Immediate amputation was performed, the patient being given 30 c.c. of antitoxic serum intravenously on the operating table. The limb was amputated at the lower third of the thigh. The wound was dressed with di-chloramin-T in oil (6.5 per cent.). The patient continued toxic, with fever of 102°. On November 11 he was given another dose of 30 c.c. of antitoxin intravenously and 60 c.c. were given into the muscles of the thigh above the wound. After this injection the patient developed a severe anaphylactic reaction. His toxemia became deeper, and although his wound cleaned up bacilli continued to be present in the smears. On November 20 the di-chloramin-T was replaced by instillations of 2 per cent. chloramin in water. The patient continued toxic, however, and died November 22. Postmortem examination was not allowed. In this case the severe anaphylactic shock prevented our repeating the use of the serum.

Case III.—St. Agnes's Hospital, No. 9463. The patient was a white boy, aged eleven years, who was admitted November 17, 1917, five days after he had received a compound fracture of both bones of the forearm near the wrist-joint. On admission it was noted that the patient was quite toxic, temperature 103°, pulse 136. There was gangrene and crackling in the wound, and smears showed the Welch bacillus. The patient was anesthetized with nitrous oxide and the wound opened up. At the same time a dose of 30 c.c. of antitoxin was given into a vein in the other arm. Exploration of the region of the fracture showed gangrene involving the pronator quadratus and the tendons of the deep flexors. Opening up the forearm near the elbow revealed gas and gangrene along the course of the interosseous vessels. There was also gangrene at the attachment of the muscles to the internal condyle. Circular amputation was therefore done at the middle of the humerus. The vessels were ligated with chromic catgut. Carrel-Dakin disinfection was begun at once. The patient improved rapidly, and on the twentieth day the protruding bone was sawed off and the stump closed by sutures. The wound then healed and the patient left the hospital a week after the closure.

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THE TECHNIC OF MAKING CELLULOID SPLINTS

By A. R. Shands, M.D., F.A.C.S.
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The making of orthopedic braces is often left to the instrument maker, which means a divided responsibility between the physician and the mechanic, with detriment to the innocent patient. The above statement, of course, has reference to the general surgeon and general practitioner and not to the careful and skilful orthopedic surgeon, who always supervises the construction of his braces and assumes all responsibility for the appropriateness and fit of the brace ordered. There is nothing more reprehensible than for a physician to refer a crippled child in need of a mechanical support for some physical disability, such as a partially paralyzed limb from anteropoliomyelitis, a distorted spine from Pott's disease or scoliosis, to an instrument maker who knows absolutely nothing of the real cause of the deformity, he being only a skilled mechanic. I am sorry to have to say that it is almost an every-day occurrence for the orthopedic surgeon to observe just such cases—many cases in which the use of any kind of a brace is absolutely contra-indicated and many in which the brace that has been applied by the mechanic is utterly inappropriate and useless, frequently worse than useless, as it is often doing harm in many ways. A large number of these patients go direct to the instrument maker without the advice of any physician, and he applies a brace that he knows of as being used by some orthopedic

surgeon, thereby taking upon himself the responsibility of a surgeon. The orthopedic instrument maker should bear exactly the same relation to the surgeon that the druggist does to the physician who writes a prescription for him to fill. Again, many braces are ordered by mail by the patient as advertised in a large number of the popular magazines. I come in contact with many such cases that are perfectly ridiculous both as to the misfit and inappropriateness of the brace.

I have almost become independent of the instrument maker since I have been having made in my office the celluloid braces which are the subject of this short paper. My success and great satisfaction in the use of them is my excuse for bringing the subject to the attention of this Association. The simplicity of making them is such that any intelligent office nurse can make them after a little instruction.

The most important part of the whole process is the making of the model over which the splint is made, and this should always be done by the surgeon. The celluloid brace has the great recommendation of being perfectly sanitary, as it does not absorb moisture, nor does it collect dirt, and is very durable. The making of them does not require the services of a mechanic, as there is no steel to be forged or straps and buckles to be adjusted, which makes the surgeon independent of the instrument maker—a very important matter in the smaller cities where there is none.

The largest field of usefulness for the celluloid splint is in spinal cases, but it has been of great service to me in the treatment of fractures of the arm and leg, especially in the treatment of Colles's fractures, as the splint can be made to give almost perfect use of the fingers, and as it is easily removed, the use of massage is much facilitated. The arm and leg splints can be carried in stock and taken from one patient to another. As the splint can be made quite flexible, almost a perfect fit can be obtained in different patients with arms or legs anywhere near the same size by means of cotton batting

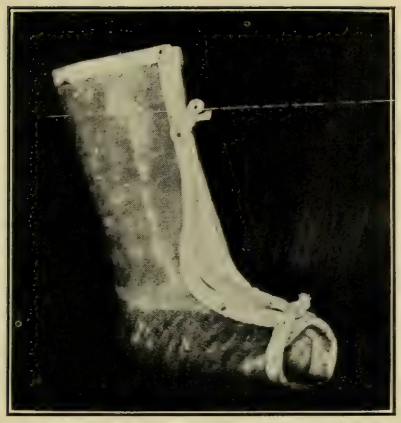

Fig. 1.—Celluloid boot for infant's club-foot.

Fig. 2.—Celluloid splint for Colles's fracture (front view).

Fig. 3.—Celluloid splint for Colles's splint (back view).

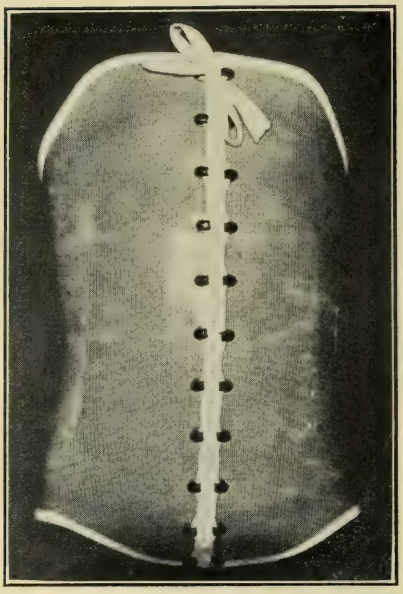

Fig. 4.—Celluloid corset for Pott's disease.

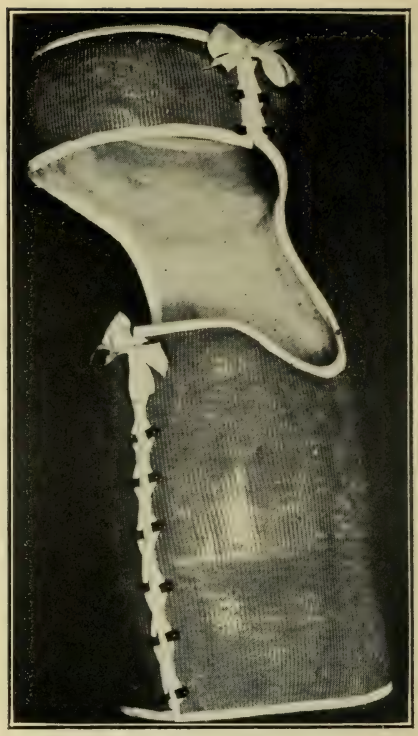

Fig. 5.—Celluloid corset for Pott's disease, requiring a head support.

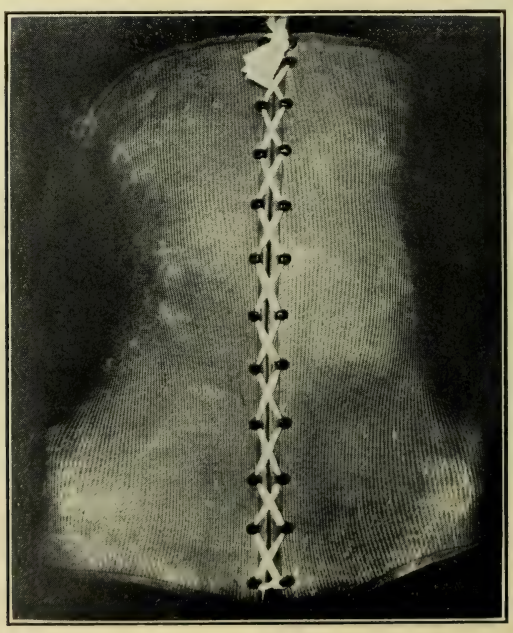

Fig. 6.—Celluloid corset for scoliosis.

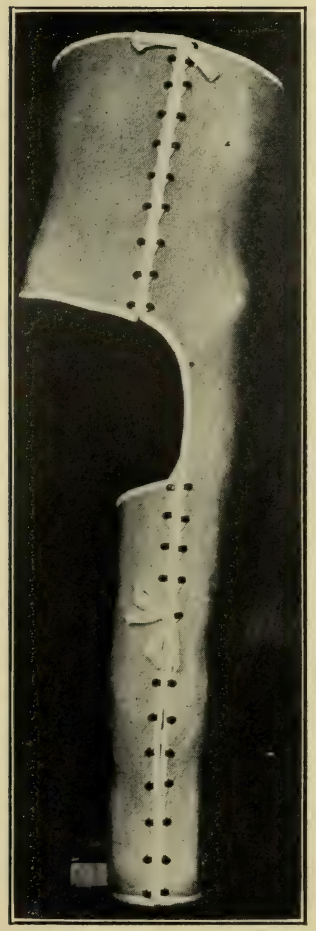

Fig. 7.—Celluloid spica splint for treatment of hip disease.

under the splint. The pressure and support to the limb can be perfectly regulated by the laces when the splint is at all flexible.

THE TECHNIC. Taking, as an example, a spinal corset, you first make a plaster-of-Paris cast of the body of the patient while suspended in the ordinary jury-mast. This cast is sealed by applying a wet plaster-of-Paris bandage around the outside. After the cast has dried, which can be done in a few hours by putting it on a radiator or near a stove, it should be well oiled on the inside with an ordinary paint brush. It is then placed on the end in a flat pan and a little plaster-of-Paris batter is applied around the bottom on the outside to seal the bottom. The cast, or mold of the body, is then filled with plaster-of-Paris batter mixed quite thin, so as to get a keen impression of the body on the torso. The torso should be cut away where special pressure is desired and built up where greater prominence is needed, as over the female breast. It is desirable to have the torso as smooth as possible, and this is obtained by rubbing it well with a wet towel while the plaster of Paris is still soft. The next step is to apply a wet gauze bandage smoothly over the torso; this is done to prevent the plaster of Paris from sticking to the celluloid corset when it is removed; this gauze bandage will stick to the inside of the corset and should be peeled off while damp. As soon as the gauze bandage is dry a gauze undervest, or a piece of tubular cotton webbing, is applied over the gauze-covered torso.

The celluloid is dissolved in acetone. A wide-mouth earthen vessel is the most convenient container for the solution. The celluloid should be dissolved in the proportion of one pound of scrap celluloid to three gallons of acetone, and should be stirred until about the consistency of paint ready for application. As a matter of economy only about as much as is needed for immediate use should be mixed, as the acetone evaporates very rapidly. The best brush for applying the liquid celluloid is a rubberset, best quality, white bristle brush, and this should be about three inches wide for the

large splints. The liquid celluloid should now be painted on with gentle strokes until the meshes of the fabric are well filled, which will take five or six applications, made at intervals of ten to fifteen minutes; this gives each coating time to dry out. If this precaution is not observed the celluloid will blister and make a rough surface. The second layer of the cotton fabric should be applied after the first application of celluloid has dried out well, which will take several hours. The painting process is now repeated as above described after each layer of the cotton fabric. The number of layers will be regulated by the desired strength. If the tubular webbing is used, from three to five layers will be needed, but if the thin gauze shirts are employed, from five to ten will be necessary. After the application of the last layer of the fabric, ten to fifteen coats of the liquid celluloid will be necessary to give the finished corset a nice glossy finish.

The corset should remain on the torso for at least two days before being removed to apply the lace hooks and binding. If it is removed before it is thoroughly seasoned it will warp and lose its shape. It should be reapplied to the torso after the hooks are set and the binding put on, and should be laced tightly and remain on for at least two days more; if this is done it will always retain its shape. It takes about one week to complete the process. The binding on the edges should be strips of a light-weight kid cut about three-quarters of an inch wide. The best possible glue for applying the kid is the liquid celluloid above described.

The same technic is followed in making the splints for the arm and leg, except that thin white stocking should be used instead of the tubular webbing and not more than two or three layers of the fabric applied, as it is desirable to have the splint quite flexible.

The lace hooks used are the same that shoemakers use for lacing shoes, and can be applied by punching holes through the celluloid about one-half inch from the border and clamping them with the same instrument employed for the purpose by shoemakers.

FRACTURE OF THE LONG BONES: A CLINICAL STUDY

By Francis Reder, M.D.
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The clinical deductions chronicled in this paper are gleaned from a case that in every aspect of bone surgery presented a very interesting picture. Before stating the record of events it must be said in justice to the surgeons who at different times exercised their skill in the hope of attaining a successful issue that they were acutely aroused by the serious task undertaken by them.

On June 2, 1912, a female, aged twenty-four years, in excellent health and splendid physical condition, was injured in a street-car collision. She was taken to the City Hospital. Examination at that institution disclosed a simple oblique fracture of the right femur about three inches below the great trochanter and a compound fracture of the left tibia and fibula. The mandible at both angles was also fractured. Under the direct care of the visiting surgeon the fracture of the right femur was reduced and the limb suspended in a Hodgen splint. This splint ensured both traction and countertraction.

The fracture of the left tibia and fibula was reduced and the leg placed in a wire splint. Three weeks later the satisfactory progress of the wound permitted the application of a plaster-of-Paris cast to the left leg.

August 2, eight weeks after the accident, the Hodgen splint was discarded and a plaster-of-Paris cast substituted.

August 9 the patient left the hospital and went home.

October 1, four months after the accident, the casts were removed from the right thigh and the left leg. Upon examination it was found there was no union of the femur nor of the tibia. The fibula was united. A consultation of two surgeons and an orthopedist was called. An open operation was decided upon. This was performed October 26, 1912. The operation consisted in refreshing the bone ends of the broken femur and securing apposition with silver wire. The wound became infected. This infective process extended over several months and severely taxed the health of the patient. The healing of the wound left the bones ununited.

Another consultation with two different surgeons was called. After an examination it was decided to break up the fibrous union that had taken place in the femoral fracture and place the limb in wooden splints, with extension. This was done March 8, 1913, in the patient's home. The splints were removed nine weeks later. No union.

May 19, 1913, at the Lutheran Hospital, a resection of femoral bone ends was undertaken and the limb immobilized in a plaster-of-Paris cast, with strong abduction. Twelve weeks later no union had taken place. The patient was sent home. Consulting another surgeon the patient was advised to go to Washington University Hospital. At that institution she was operated on October 13, 1913, sixteen months after receiving the injury. A graft from the right tibia was transplanted into the ununited left tibia. The leg was encased in a plaster-of-Paris cast. December 29, 1913 (ten weeks later), it was removed and union was found to have taken place. A cast was reapplied.

January 14, 1914, a graft from the right tibia was transplanted into the ununited right femur. The graft was of the inlay variety. At the time of the operation no undue attempt was made to correct the deformity. The surgeon's sole aim was to excite an osteogenetic force sufficient to accomplish a bony union. This accomplished

it was reasoned that the correction of the deformity could be brought about by a subsequent operation. Inasmuch as the adductor group of muscles was very much contracted at the time of the operation, this was a logical procedure, as it would have been impossible, even with the best of fixation, to prevent a possible accident to the graft. This was clearly demonstrated to me in the subsequent course of the case. Furthermore, the surgeon, being alert to the defective osteogenetic power of the bone ends, decided not to force the limb into any absolute fixation, but simply to allow it to rest in a comfortable position, so that no compression or constriction could exert any inhibitive influence on the callous formation. At times the contractions were sufficiently violent to endanger the graft. The patient begged for slight traction on her limb that the muscle spasms might be to some extent controlled. Traction of about ten pounds was applied. A certain amount of relief was thereby obtained.

When she left the hospital on March 1, 1914, six weeks later, union with a greater deformity had taken place.

May 11, after being at home a little over nine weeks, she returned to the Washington University Hospital, where the cast from her left leg was removed. The union of the tibia was found firm, but there was a marked posterior bowing. The patient remained at the hospital seven weeks for after-treatment, and was then sent home.

September 1. The patient returned to the hospital for treatment for her knee- and ankle-joints and *x*-ray examinations. She remained three weeks. From September 21, 1914, to July, 1915, the patient was at home. There she was subjected to massage and motion exercises in a most assiduous manner.

July 15, 1915, she returned to Washington University Hospital to have her knees and ankle-joints, which were partially ankylosed, manipulated under an anesthetic. She remained fifteen days. From June 2, 1912, to July 15, 1915, the patient had been subjected to fifteen ether and five gas-

oxygen anesthesias. The time from August, 1915, to February, 1917, was spent by the patient at home, applying the various motion exercises she had learned while in the hospital and having her limbs massaged. She made every endeavor to learn to walk, but did not seem to be able to make any progress, and, as she expressed herself, her muscles absolutely refused to act right. She complained of much pain in her back, principally the lumbar region. The fracture sites, too, were at times very painful.

February 7, 1917, I was asked to interest myself in her behalf. The patient stated that measures which in the past were helpful had now reached their limitations. She was unable to improve on her present state of walking and the future was beginning to grow dark for her. It was a request that could not be denied. The physical examination revealed the patient in a fairly good condition. This was remarkable when the experience of the last four and a half years of her life was considered. From the want of proper exercise a general anemia was contracted, and this was very apparent. However, she expressed herself as feeling in good health. With this expression there was linked an asset, very essential for the sustenance of hope, willingness and fortitude. When the patient was asked to walk it was with the greatest effort that her lower extremities were placed in motion. The contortions of her body resembled those of a tabetic. A heel three inches in height was necessary on her right shoe, while her left leg had to be supported by a brace.

Examination of the right lower extremity revealed a shortening of three and a half inches, with marked inward rotation. The knee- and ankle-joints gave about one-fourth normal motion. In the hip-joint motion was quite normal. A deformity in the upper third of the thigh, such as would result from angulation and outward bowing of the femur, was apparent. The amount of callous formation about the fracture site was very large. The union was very firm. Atrophic changes in the musculature of the limb were

marked. The group of adductor muscles was contracted. On the outer side of the thigh the skin was adherent to the bone, a marked depression interrupting the normal contour.

Examination of the left leg revealed an extensive bowing of the tibia backward. Callous formation over the site of fracture, which was in the middle third, was rather extensive. The union was firm. From the cicatrix in the middle third of the leg it was evident there was much destruction of the soft tissues. The foot was in a "drop" position, with an extremely limited motion of the ankle-joint. The knee-joint gave about one-third normal motion. Motion of hip-joint was normal.

Examination of the pelvis elicited a marked obliquity, compensatory to the shortening of the right leg. The spine gave evidence of lateral curving to the left. The lumbar muscles on the left side of the spinal column were prominent. Here was a terminal condition which, after a period of four and a half years in a patient who had been subjected to the skill of very competent surgeons, proved most disappointing. Everything that was good and effective in both the closed and open methods of treatment was employed, and yet the end-results were poor.

The condition of the patient presented many problems, and it became a question with me to what extent they could be worked out. Why this patient did not have a primary union of the right femur must be explained upon the ground that after the broken bone ends had been brought in good alignment, the proper fixation of the fracture, through some cause or other, was not maintained. In the left tibial fracture a septic process interfered with union. Later, when the right femur was subjected to the open method of treatment, it was also a septic process which pervented union. Seven months later, when the bone ends of the ununited femur were resected and no union resulted, it must be inferred that the osteogenetic forces were defective or that the fixation of the limb was inefficient, permitting a material amount of movement to take place between the fragments sufficient to interfere with the process of consolidation.

The introduction of the bone graft clearly demonstrated it was a direct assistance to osteogenesis, both the fractured femur and tibia giving the evidence by promptly uniting.

The success of the bone graft lends an interesting chapter in this case. Almost two years after failure of union the introduction of an autogenous bone graft caused the bone cells to respond to the stresses to which the part was subjected. Consolidation took place. That the osteogenetic forces were weak and that the callus produced was not sufficiently strong in its consolidation to permit weight-bearing became apparent through the resulting deformity in both femur and tibia. It would appear that five months (femur) and seven months (tibia) might be sufficient time for the consolidation to support the body weight without disaster. There is no definite time when it can be said that a bone whose continuity had been reëstablished through the stimulus of a bone graft will be sufficiently strong to bear the body weight without dire consequences. It is fairly safe, however, to assume that a grafted bone will require at least three times as long for a firm consolidation as a broken bone which has responded to primary union.

Having fully acquainted myself with the clinical manifestations presented in this case a method of procedure was decided upon.

From the skiagraph it became evident that the three and one-half inches' shortening of the right femur was permanent, *i. e.*, an attempt to lengthen the limb through operative means would be futile and that the correction of the angulation would not add materially to the length of the bone. To equalize the difference in the length of the limbs it was decided to excise three inches of the left femur. With this procedure it was expected to restore to a certain extent the existing obliquity of the pelvis and to aid in the obliteration of the lateral lumbar curvature. Furthermore, it was expected that this procedure would more readily overcome the unequal strain that had been thrown upon the joints and ligament

through the faulty transmission of the body weight upon the lower limbs. The correction of the right femur was brought about by removing with a chisel a wedge from the convex side of the angulated bone. This was made imperative because of the hard and extensive callous formation. The callus was found uniform in its density throughout. A piece of the graft, about one inch long by one-half inch in width, having the appearance and characteristics of a sequestrum, was removed. Before the femur could be brought in proper alignment it became necessary to chisel through its entire thickness. Recalling the various pathological stages this bone had passed through, and being fearful that a latent septic condition might be stimulated into activity, the bone and soft tissues were subjected to the least amount of traumatism possible. It is well to remember that organisms remain quiescent for a long time in a fracture which has united firmly and apparently normally after an infection, and that a subsequent trauma or the introduction of foreign matter, such as plates and screws, may stimulate them into active growth and so endanger a junction.

To guard against any displacement the bone ends were secured with a bronze wire. The wire was given preference over a Lane plate because of the fear of inciting a possible infection and, furthermore, because experience had proved that a plate can produce actual delay in efficient osseous union. Inasmuch as it could be assumed that in this extensive callous formation the osteogenetic force was deficient the contra-indication to the plate became the more apparent.

Blood bathed the exposed bone freely. Closure was made without drainage. This was done under the assumption that the presence of blood about the traumatized bone might be of direct assistance to osteogenesis. To further facilitate the bone production the thigh was subjected to a congestive treatment, the so-called damming, originated by Thomas, which has served me well in several cases of delayed union. The limb was corded with a rubber tube applied above and

below the fracture, sufficiently tight to produce considerable swelling and stasis, for several hours a day. A Liston splint reaching to the axilla was applied to the extremity. The fixation seemed to be satisfactory. So long as the patient was under the influence of an anesthetic the group of adductor muscles, which prior to the anesthesia were markedly contracted, remained relaxed and the splint did not betray its shortcomings. However, after the effect of the anesthetic had subsided the reflex contraction of the muscles manifested itself, disturbing the fracture and causing great pain. A Buck's extension was applied in the hope that gentle traction would overcome these muscle contractions. No difference, however, could be noted. Suspecting that the damming could excite or aggravate the spasms it was discontinued. It was, however, found that cording of the thigh lessened their severity and the reapplication of the rubber tubing was resorted to for even a longer period of time.

After four weeks, during which time the patient received an opiate almost daily on account of the pain caused by the muscle spasms, it was decided to encase the limb in a plaster-of-Paris cast in the hope of getting more efficient support through a firmer fixation. The cast enveloped the lower part of the trunk and extended up to the thorax. It encased the whole extremity, including the foot.

The change seemed to be conducive of some good, and although the spasms did not subside entirely they became less severe and of shorter duration, with longer intermissions. On account of atrophic changes in the limb, repeated applications of casts became necessary. Before a new cast was applied the limb was not encumbered with any form of splints except a sand-bag support for two days. During this time the whole extremity was bathed and massaged, and the articulations subjected to passive motion. It was interesting to note how quickly the limb changed from its blanched color to a livid one after the cast was removed, giving distinct expression of how restricted the blood supply must have

Fig. 1.—Angulation of right femur with firm union following inlay bone graft for ununited fracture. There is about $3\frac{1}{2}$ inches shortening. The valgoid condition of the right leg is the resultant weakening caused by excision of bone to be used as grafts for the right femur and the left leg.

Fig. 2.—Backward bowing of left tibia with firm union following inlay bone graft for ununited fracture.

Fig. 3.—Correction of angulation in right femur. Firm union.

Fig. 4.—Correction of backward bowing of left tibia. Firm union. An excision of three inches of the left femur was made to equalize it with its fellow and to relieve the pelvic obliquity and the lateral curvature of the spine.

been to the parts. Such a picture as this strengthens the belief that a plaster-of-Paris cast is not infrequently the cause of a delayed union, but may be a potent factor in non-union. The application of a plaster-of-Paris cast to a broken bone must be a studied procedure. Its shortcomings must be well understood and its indiscriminate use should be discouraged. A plaster cast must not be applied to be constrictive or to compress the limb, but must be restrictive only, fitting closely to its actual somewhat increased circumference and forbidding further increase of this circumference by muscular effort. At the end of three months the plaster cast was discontinued and a wooden support of posterior and lateral splints was substituted. The muscle spasms had not entirely ceased. At times they were very severe. However, the contractions became less frequent, their favorite time being in the morning, in the evening or at midnight. During the first week in September, seven months after operation, a test of the limb revealed a firm union and all splints were discarded. Mild muscle spasms still supervened. The patient was not yet permitted to leave her bed.

The operative problem of the left femur presented difficulties entirely different from the right. To equalize its length with its fellow an excision of three inches was to be made. Could this excision be made without interfering to an appreciable extent with the function of the limb? Would it be possible for the muscles to contract so as to have coördinative utility and be able to accommodate their sense to a degree that would ensure an almost normal muscle balance? Taking into consideration that this patient was fully matured, these questions possessed a weighty import. Inasmuch as fractures of the femoral shaft resulting in two inches, shortening terminated in satisfactory function, it was reasoned that an additional inch would not prove a serious menace. The excision was made at the time when the deformity in the right femur was corrected February 19, and the site chosen was that presenting the best mechanical advantage. This seemed

to be in the middle third immediately above the nutrient foramen. With a Gigli saw the excision was accomplished with ease. When the bone ends were approximated the muscle structure sagged about the bone in large masses. It was not a promising picture. To retain the bone ends in apposition and prevent a possible displacement a graft taken from the excised bone was pushed into the medullary canal of the femur. A parting of the bone ends was anticipated by introducing a bronze wire through the upper and lower fragment. This bronze wire was tied in a knot as an ordinary piece of twine. At the conclusion of the operation everything appeared satisfactory and the limb was immobilized with a Liston splint. Four hours after the effects of the anesthetic had passed off there occurred violent muscle contractions. They were irregularly intermittent and shook the limb. It was necessary to administer an opiate to relieve both muscle spasm and the pain every ten to fourteen hours. On the fifth day after the operation a skiagram revealed that the intramedullary plug was forced out, that the wire had given way at the knot and was now an elongated loop and that the bone ends had parted to the extent of about two and one-half inches. The damage which the violent muscle contractions had wrought was a great surprise, and inasmuch as they did not abate in their severity, the only alternative was in the application of a Lane plate.

After removing the wire and the intramedullary plug which was found lying outside of the medullary canal a heavy plate with six holes was placed without difficulty. The junction was found strong after four screws, two in the upper and two in the lower fragment, had been introduced; for this reason the remaining two screws were omitted. The limb was encased in a plaster cast in an abducted position. This cast, like the one on the right limb, included the foot and extended up to the thorax. To anticipate muscle spasms after the effects of the anesthetic had passed off an opiate was administered. As long as this patient was under the influence of an opiate

she was free from these painful contractions. Upon withdrawal of the drug the muscles, principally the adductor group, would contract in a violent manner, causing great pain and endangering the junction.

On the eighteenth day after the plate had been introduced the patient informed me that during the night the muscular contractions had been so violent that she feared the plate had been broken, as she distinctly heard a snap coming from that part of her leg. An *x*-ray picture taken immediately revealed the plate not broken but the screws in the upper portion forced. They could be plainly seen protruding above the plate. The alignment of the bone was only fair, the lower fragment having been drawn inward, causing a slight angulation at the junction.

The condition was disheartening. To again open the wound and correct the existing deformity by reapplying the plate would have been a justifiable procedure. However, it was reasoned that the existing condition did not warrant it. Manipulation of the limb gave assurance that the plate was still fixing the bone ends quite securely. It was therefore decided to place both limbs upon a Rainey frame, and when the time came for removing the plate, then to make such corrections of the deformity as were deemed advisable.

June 18 the plate was removed, 114 days after it was introduced. The condition found was about the same as revealed by the *x*-ray picture taken when the plate was supposed to have been broken. The screws in the upper fragment were out of their holes. The screws in the lower fragment were loose and could be removed with ease. Angulation of the bone was quite marked, the lower fragment having been pulled perceptibly inward. The Rainey frame undoubtedly was of great service in preventing a greater deformity. The behavior of this fracture clearly demonstrated to me how a non-union in the right femur resulted during the early measures that were instituted.

Although the position of the bone ends was fairly good it was found that after nearly four months the callus was tender and no difficulty was experienced in readjusting the bone into the proper anatomical line. It is a rule in fractures that the worse the position of the bones the longer will the callus take to consolidate. In this case, however, although the apposition was not good, the limited amount and soft condition of the callus cannot wholly be ascribed to the poor apposition, but must be attributed to some extent to the anemic state of the patient. The limb was again encased in a plaster-of-Paris cast and placed in a Rainey frame. Muscular contractions became more mild and only occasionally was there a marked exacerbation.

July 20, at 2.30 P.M. (five months after the operation), a muscular spasm of great severity was experienced which lasted three hours. This was the last of the severe contractions. An opiate was now only occasionally necessary. The inference could readily be made that this patient had become an habitué, having received almost daily doses for three months and triweekly for two months. However, it was with comparative ease that the discontinuance of the drug was effected. This was indeed remarkable. During the last two months of her stay in the hospital no opiates were administered. The muscular contractions continued for seven months; only during the last two months were they so mild that no notice was taken of them. Antispastic remedies and quite a few mechanical measures to overcome these muscular spasms that so seriously threatened a good end-result did not influence the condition. Morphin was the only remedy that gave relief. It was hoped that the muscles would give up the struggle and acquiesce, as had been my experience in most cases of fracture where such contractions were severe enough to attract attention.

In this case, however, the phenomenon presented itself in such an unusual form that a central lesion was being suspected. A neurologist, however, assured me that no

such lesion was present. Inasmuch as temperature and pulse remained within a range that would preclude any pathological changes in or about the fracture during the process of consolidation, the persistency of these muscular spasms must be ascribed to an asthenic condition of the patient, inviting a neurological inanition, where a trauma so agitated the nerve centers that the coördinative powers of the muscular apparatus gave evidence of disconcerted action, and continued to do so until more favorable nutritive changes had taken place in the system. At no time did the temperature rise above 100° nor the pulse- rate exceed 90, excepting on August 2, when the temperature registered 102°, with a pulse rate of 118. The sudden change in the temperature and pulse picture was caused by an acute attack of appendicitis. The appendix was removed the following day. It showed evidence of marked thrombotic changes. Five days later temperature and pulse were again within the normal range.

September 4, when the plaster encasement was removed from the left limb, muscle atrophy was marked. The contour of the thigh, however, was good and the firmness of the muscles presaged a good functional result. In this I was not disappointed, as the patient is not experiencing the difficulty in walking that had been expected. The greatest hindrance to overcome now is the partially ankylosed condition of both knees and ankle-joints. These articulations, although close attention was given them, suffered much during five years of confinement.

How near these articulations can be restored to their normal action is problematic. At present it is a serious handicap to the patient's walking exercises. Too much stress cannot be placed upon the care of the articulations in fracture of the long bones. It does not matter much whether the fracture is in the immediate vicinity of the joint or not. The neglect of extending the needed attention to a joint of a limb that has been immobilized is almost a surgical sin. No matter how serious the bone injury there is always an oppor-

tunity at some time when the proper consideration given a joint may avert much that is aggravating and frequently very troublesome. Especially is this true in fractures involving the long bones of the lower extremities.

The remaining deformity that necessitated correction was the bowing backward of the left tibia. The amount of callus about the middle third of the tibia, the site of the fracture, was abundant. Consolidation was strong. It will be remembered this was a compound fracture, the infective process covering nearly three months. A non-union resulted. This ununited fracture was subsequently stimulated into union with the assistance of a bone graft taken from the right tibia. The deformity was undoubtedly caused by too early weight bearing, when the callus was the seat of active changes and consolidation was far from complete.

In correcting this deformity, which was undertaken the same time the right and the left femur were operated on, an oblique osteotomy was done through the callus between the original fragments of the shaft. The tibia was not severed in its entirety, enough of the bone being kept intact to act as a splint for fixation to prevent any overriding and subsequent shortening. After the chisel had penetrated the tibia to about four-fifths of its thickness the lower fragment was forced backward, the action being similar to the correction of a green-stick fracture, until the posterior bowing had been overcome. The result of this manipulation left a gap about an inch wide upon the anterior aspect of the tibia. This gap was allowed to fill with blood and the skin closed over it. No trace of the bone graft was found. With posterior and lateral splints the leg was securely fixed and bandaged to the Liston splint, which was used to immobilize the thigh. Subsequently, when the Liston splint had to be changed on account of the muscular contractions, the entire extremity was encased in a plaster-of-Paris cast.

The anatomical line of the tibial shaft remained as it was placed at the time of operation and a good consolidation resulted at the end of three months.

This patient is at present able to stand erect and is making satisfactory progress in walking. Muscle fatigue and joint pains necessitate that the exercises be of short duration.

The long confinement caused ligamentous relaxation in nearly all the large joints of her body. This was exemplified in the frequent subluxations of her shoulder-joints by some trivial movement.

In summarizing it may be said that the dominant facts in this case were:

The successful bone-grafting sixteen to nineteen months respectively after the fractures had been incurred.

The successful bone-grafting following a septic process.

The successful bone-grafting after non-union following resection of the bone ends.

Excision of three inches from the femoral shaft, without causing any apparent damage to the mechanics of the thigh muscles.

The violent and long-continued (seven months) muscular contractions.

Failure to anticipate these muscular contractions.

Failure to judge the violence of these contractions.

Failure to properly secure the bone ends of the left femur after excision.

Failure to properly immobilize the extremities from the start.

The successful molding of the callus of the left femur four months after excision.

The length of time it required for the consolidation of the callus to permit weight-bearing.

The promptness with which the patient was able to discontinue the morphin.

BONE GRAFT AND ARTHRODESIS IN RECONSTRUCTION SURGERY

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The war has given an impetus to "reconstruction surgery." It has been suggested by the Army Medical Department that civil surgeons begin to "round up" the crippled and deformed in and among their clientèle and such surgery be advised and done on them as will tend to place them in a functionally more useful condition.

This is a "preparedness propaganda," conserving the resources of the State in increasing the efficiency of every deformed individual and fitting the surgeon to meet a probable demand for restoration to function of those maimed in the fight for universal freedom. No greater source of enthusiasm than one's patriotic desire to do his part is needed to meet this demand among those of us whom circumstances has kept at home, and it is hoped it will so perfect our skill and technic that we will not fail.

Orthopedic surgery affords a fertile field for useful work. This branch has not been attractive to the majority of surgeons for reasons which arise from two different points of view held by patient and surgeon: the former expects an absolute anatomical, functional cosmetic restoration with an immediate return to normal appearance and usefulness, the latter hopes primarily to reëstablish functional usefulness and along with it such anatomical and cosmetic results as

are compatible with the maximum degree of serviceability, and is satisfied with ultimate and partial restitution. The result is a dissatisfied though benefited patient and a doctor chagrined with disappointment. The psychology of enthusiastic endeavor depends on more than the factor of self-satisfaction; the pleased patient is no small incentive in this matter. It is an essential safeguard that a none too optimistic outcome is pictured to the patient; better obtain benefiting results beyond the promise than to fall below an assured expectation.

There is a tediousness or time-taking element in reconstruction surgery greater than in ordinary operations; particularly is the contrast noticeable when compared with abdominal surgery, especially that done for acute conditions both in operation and subsequent course of after-treatment. There is a pain habit existing as truly as the status epilepticus, and painful distortions endured over many years require a long interval of time after operative relief before the pain impulse is obliterated, as discouraging to the patient as it is irritating to the surgeon.

It is not the purpose of this paper to demonstrate any new technic but to emphasize some of the fundamental factors making for success, with reference to apparatus and appliances, as well as certain precautions apparently or evidently essential before, during and after operating, and to make a plea for early operation in destructive joint disease, in metastatic osteoarthritis, doing radical arthrodesis before extensive destruction of joint structures or bony framework has occurred. In the light of more extended experience it may be predicted that delay in operating on infected joints of certain selected types may soon become as reprehensible as that of delay in operating for osteomyelitis.

Preliminary preparation of the patient in building up resistance with vaccines, protecting with sera, elimination of intestinal toxins, alkalinizing the system, loading it with carbohydrates in the form of glucose and filling the tissues

with fluids before and in prolonged operations by hypodermoclysis during the operation are all well understood—in fact, commonplace—yet need constant emphasis not only because they are elements of success in the desperate cases, but they also add a world of postoperative comfort in every case when judiciously employed.

The equipment essential for the best results in arthrodesis and bonegraft surgery, unless the surgeon has a mechanical turn or talent, must be extensive as well as expensive. I shall demonstrate by models and photographs a practical working basis in equipment which is both inexpensive and efficient. The table here shown, when to it is added the portable attachment, will at a small cost do all that the more elaborate and expensive tables will do. This modified Bradford frame is really a product of the University of Pennsylvania Hospital simplified. The frame can be made in any plumbing shop. It has the splendid advantage of permitting the patient to be moved in bed for bathing and for defecation without pain or disturbing in any way traction, extension or position of the parts and of retaining them in exact apposition during any desired changing of the patient, who can be moved out on porch or in sun parlor with ease, as frame and patient are all one piece. Among other advantages the frame and canvas can be placed over either the orthopedic or operating table and the adjustments made during or on completion of operation, fixed and retained before the patient is moved. In some instances this is an important factor, particularly in arthrodesis and in certain fractures with insecure fixation by bone graft.

A simple extension apparatus is made by inserting sash pulleys into a pine board; for extension the board is placed at the foot of the bed and secured by cords. For an overhead trolley a long piece of timber, 2 x 2, with screw pulleys at intervals, is supported over the bed on four pieces, two at the foot and two at the head of the bed. Various wire, angle iron, tubular frames or splints can be made at any black-

smith shop to suit the case in hand. These are real tailor-made splints and are vastly superior to the stock pieces advertised or sold by instrument houses; particularly is this true of the Thomas and other wire splints in general war use. Add to this plaster-of-Paris bandages, with a few straps and buckles, and a definite knowledge of what one wishes to accomplish, and the armentarium for a working basis is complete.

Technically the joint, bone and tendon operations are not more difficult, but the successful outcome is more largely dependent on absolute asepsis; then the chances are relatively far greater for failure following a careless or indifferent aseptic technic in this class of surgery than in any other. Infection not only spells failure, but frequently is life-taking. It almost certainly deprives the patient of future opportunity for a second successful trial. This is the real domain of knife-and-fork-surgery.

Antiseptic treatment is a preoperative measure, while aseptic technic is paramount and must be actuated by mental alertness, with a regard for strict cleanliness approaching fanaticism, with hands trained to avoid touching the open wound, to lose for once in all this class of work that wealth of information gathered from the educated touch of exploring deeper or unseen recesses, as is so valuable in the course of abdominal operations. Experience in time will soon give the same impulses, though diminished in fineness of perception, through the grasp of the handle of knife, chisel, gouge or elevator. Incised margins of skin should be protected and no skin left exposed after incision. Every instrument regarded as unfit for further use once it is removed from the wound until it has been boiled; discarding each gauze mop from its metal holder as soon as it has wiped the wound one time only; regarding the gloved hands as a menace if they touch not only the wound but also the part of the instrument which will touch the tissues; handling thread and needles with instruments only. Every knot can be as securely tied

with hemostats as with the fingers, and almost as quickly. Hemostats should be used invariably for this purpose instead of the fingers. The surgeon must not only have an aseptically trained conscience and hand, but his assistants must be equally so.

The work of Carrel, Dakin and associates, if carried out in systematic detail, is, beyond question, time and tissue saving; permitting clean work to be done in recently septic wounds within a few weeks after instituting the treatment; the microscopic findings of not more than one organism to three fields being the guide in instituting final surgical effort and a closed operation in an open discharging though clean wound. The 2 per cent. formalin glycerin injection of septic joints after the method of the late J. B. Murphy, previous to arthroditic operations, is correspondingly efficient in this class of surgery.

In the Vaughan Memorial Clinic the above preliminary treatment in cases of types indicated has been so satisfactory that I have found no occasion to try out B. I. P, or the anilin dye pastes; the latter, however, because of staining necrotic tissue in addition to microbicidal properties have a distinct field of usefulness in recently septic wounds, particularly in Bacillus capsulatus aërogenous infections, where the removal of all dead tissue is requisite to eliminate spreading cultures of this organism while free and liberal incisions permit drainage. The removal of all focal infection preliminary to bone, joint and tendon operations is a safeguard against wound-infection that should never be overlooked and neglected. Wherever and whenever time permits, teeth, tonsils, accessory sinuses and all possible sources of focal infections are removed prior to operation, otherwise these are attended to at the same time the other surgery is done.

Specialism has the disadvantage of onesidedness, exaggerating one's estimate of reflex influences or of the relationship of that specialty to the general morbidity existing in the patient under treatment, with the result that all disorders of

the system are attributed by the specialist to the pathology under treatment by him. Getting into this groove is rather difficult to avoid, because we very frequently observe distant and apparently disassociated pains, and disabilities disappear after removing some remote disease or deformity. Thus arise fads in medicine, making fashionable appendectomies, removal of teeth, tonsils, gall-bladders and such organs as are not essential to life, under the suspicion that they are harboring focal infections, causing reflex nervous or digestive disturbances.

Not to be distanced, orthosis in surgery is beginning to include in its special line of work, in addition to the gross deformities, the obscure deviations and the general morbidity possibly resulting therefrom.

Persistent efforts over long periods of time, with a remuneration far less proportionately for time and effort expended than in almost any other department of surgical endeavor, is to be anticipated. Final results that are worthy of best efforts follow efficiency raised to the nth power.

Reconstruction operations are the comsummation of conservative surgery. All is saved that can be used and all is used that can be saved.

The first three cases in this report are of metastatic osteoarthritis, representing varying degrees of destruction of the hip-joint as time lengthened and disease extended, the methods employed in each, results obtained and conclusions deducted therefrom.

Case I.—Mrs. C., white, aged thirty-six years, married. History of injury to left hip when two years old. At eleven fell and fractured left leg above knee. In bed for weeks following this injury. Recurring attacks of pain in hip. Left leg shorter. Thrown from buggy in 1916, left hip injured. Operated for appendicitis while still suffering from hip. Hip not treated after injury. Abscess pointed and drained on inner side of thigh near crotch; septic condition continued, with recurring chills. Entered the Vaughan Memorial

Hospital October 15, 1916. Sinus discharging on inner side of thigh, fluctuation under reddened area, outer aspect of thigh; septic. Pus evacuated and tube inserted in both openings. Culture showed colon bacilli predominating. Dakin-Carrel treatment begun and three weeks later discharge contained not more than one microörganism to three fields. *X*-rays showed the trochanter riding three inches above the acetabulum. Head and neck of femur completely absorbed. Upper third of femur diseased. Operation done December 18, 1916. Exposure of joint through Murphy goblet incision. Acetabulum and all diseased bone curetted. Upper third of shaft of femur tunnelled. Large pyogenic sac surrounding joint curetted. Aponeurosis of contracted muscles divided and tendons lengthened, so that firm traction was made with pulleys. End of femur brought on line with upper margin of acetabulum. Lax structures, consisting of fibrous tissue and remnants of joint capsule, were dissected free, including some underlying muscle tissue, and sutured in upper third shaft of femur, completely filling hiatus made by chisel and curetted. Divided muscles sutured with catgut and wound closed without drainage. Dressings and plaster cast applied, using canvas and frame overlying orthopedic table. Extension straps of limb were transferred to pulley of frame, thus securing desired position without moving patient. After operation patient and frame, being all one piece, were removed from operating room after all adjustments had been made. Wound healed without incident, and in three months patient left hospital, February 19, 1917, wearing a Thomas splint. She is walking with one crutch. Has movable hip-joint, with three-inch shortening of limb. Is in splendid physical health. As time goes on will be able to discard crutch. Limp being overcome by elevating shoe on that side (Fig. 1).

Case II.—Mrs. P., widow, aged sixty-two years. Came to the Vaughan Memorial Hospital May 22, 1917, to have new brace made for hip. Family history negative. Hip-

joint disease began at the age of two years following an injury. Recurring exacerbations during sixty years. General physical health had been good, except for these attacks, necessitating confinement to bed, with extension, then crutches and braces. During the acute outbreaks the disability was complete and pain agonizing. At the time of examination her temperature was 99.4° and pulse 80. Blood count: leukocytes 12,000, red cells 4,000,000, neutrophiles 76 per cent. Teeth carious, pyorrhea and recession of gums. *X*-rays of right hip showed complete bony ankylosis, fusion of neck, head and acetabulum into one bony mass. Typical adduction of deformity and emaciation of thigh and leg. Continuous pain in hip, exaggerated by every movement. Advised arthrodesis and did the Murphy operation May 29, 1917. The margin of acetabulum was entered by chisel and only spongy neck of femur remained, the head having been entirely destroyed in osteitic process. The acetabulum was curetted and reamed out. Neck of femur shaped to fit. An aponeurotic flap interposed and the operation completed step by step, as described by Murphy; used phosphor-bronze wire instead of the nail in replacing the divided tip of trochanter. Primary union was secured throughout and convalescence was in every way without incident, except for recurring attacks of pain, which occurred without any explanation except that of pain habit. She was kept on the retentive frame, with extension and counter-extension, fixation of hips in double spica plaster bandage for three months. During remaining three months in hospital she was on crutches and allowed to move the leg as in walking, with only the toe touching the floor. She can stand, sit or lie down with comfort. She has adduction, abduction, flexion and extension. The voluntary range of motion is limited by the extreme attenuation of all leg muscles from long disuse. The thigh muscles were thin and flabby, with considerable fatty degeneration. Her teeth were extracted at the time of operation. She has gained twenty pounds in weight and is in better physical health

than ever before. Dismissed from hospital December 14, 1917 (Figs. 3 and 4).

Case III.—F. J. A., male, white, aged eleven years. Admitted to the Vaughan Memorial Hospital October 3, 1917, for hip-joint disease of four years' duration. In 1913 was thrown while at play in school, falling on left hip. Continued at school that day but was unable to move the hip the following morning, and was in bed for two weeks, with stiff and painful hip. Has been lame in hip ever since, confined to bed at intervals on this account, though up most of the time. After taking horseback ride the last of September he was compelled to take his bed on October 3, 1917. Entered the hospital with temperature 99.4° and pulse 84; stiff left hip, painful on pressure and held rigid by tense muscles. Thigh adducted and leg flexed. Attempts at passive motion caused severe pain. Blood picture showed polymorphonuclear leukocytosis. *X*-rays revealed acetabular disease, with bulging of fossa. Teeth were good, tonsils enlarged and no history of sore throat. He was put to bed and extension applied. October 6 temperature reached 102° F. On the seventh day he was given mixed vaccine. October 8 his lowest temperature was 98.8°, highest 100° F. This case is of unusual interest because it presented a doubt as to the best methods to pursue in its treatment. Here is an acetabulum filled with fluid, bulging its wall into the pelvis, eroded and with a femoral head apparently uninjured; if so in its incipiency. The case is one during an acute exacerbation, with an extending rise of temperature and a leukocytosis. Under older conservative teaching this case should have been placed in bed, extension and counter-extension applied, and time would not only have caused the inflammatory process to subside, but no doubt would have resulted in a temporary cure, or the disease in its continued progress might have eroded and finally destroyed the elementary substances of the joint structure.

A resumé of Case I shows just such a condition which per-

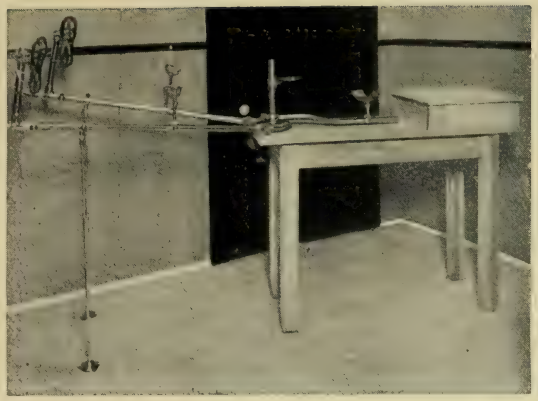


Fig. 1.—Orthopedic table, Echol's traction, fixation apparatus assembled.

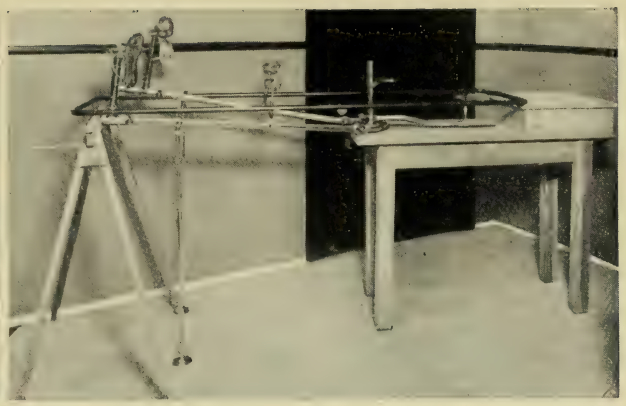


Fig. 2.—Orthopedic table with Bradford frame.

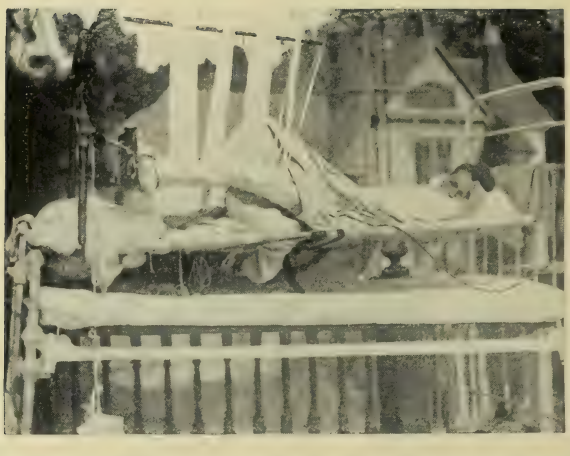


Fig. 3.—Photograph showing patient in fixation frame on bed.

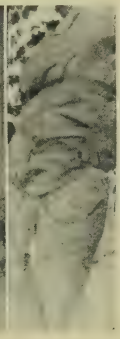


Fig. 4 Fig. 5 Fig. 6

Fig. 4.—Case I. Osteo-arthritis. Complete destruction of the head and neck of the femur. Displacement of the upper end of the femur. Osteomyelitis of the upper third of the shaft.

Fig. 5.—Case I after operation.

Fig. 6.—Case I. Photograph of hip after recovery.

Fig. 7 Fig. 8

Fig. 7.—Roentgenogram showing complete bony ankylosis between acetabulum and femur.

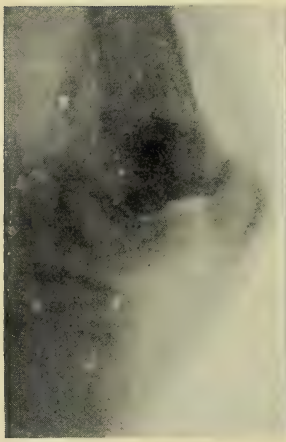
Fig. 8.—Roentgenogram showing results of arthrodesis.

Fig. 9 Fig. 10 Fig. 11

Fig. 9.—Case III. Metastatic osteo-arthritis of the hip-joint, showing acetabular disease with bulging of its walls into the peritonea cavity.

Fig. 10.—Roentgenogram showing the results of arthrodesis in Case III.

Fig. 11.—Photograph of the back in Case III, showing recovery. Case IV.



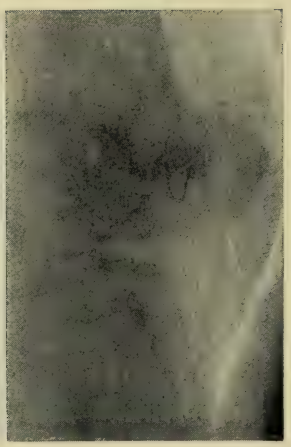


Fig. 12 Fig. 13

Fig. 12.—Bony ankylosis of the knee in epiphyseal fracture of the femur.

Fig. 13.—Roentgenogram of Case IV taken after operation.

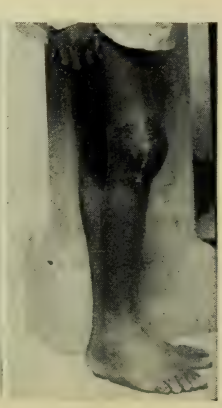


Fig. 14 Fig. 15

Fig. 14.—Photograph of Case IV showing the patient sitting after arthrodesis of the knee.

Fig. 15.—Case IV standing.

Fig. 16

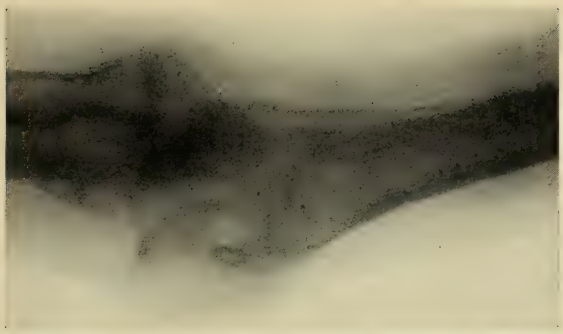


Fig. 17

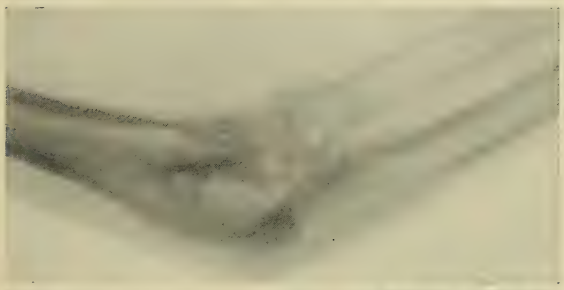


Fig. 18

Fig. 16.—Roentgenogram Case V showing anteroposterior view of fracture dislocation of the elbow-joint.

Fig. 17.—Roentgenogram Case V showing lateral view of fracture dislocation of the elbow-joint.

Fig. 18.—Roentgenogram Case V showing lateral view of elbow-joint after operation.

Fig. 19 Fig. 20

Fig. 19.—Photograph of Case V showing maximum degree of flexion of arm on forearm before operation.

Fig. 20.—Photograph of Case V showing flexion after operation.

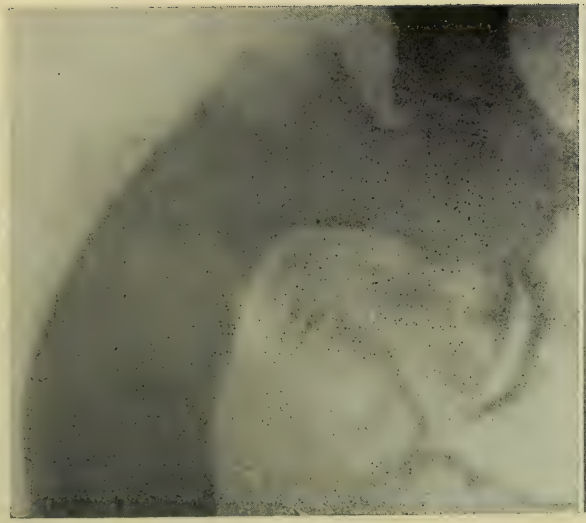


Fig. 21.—Case IV. Roentgenogram taken before operation. Osteitis cystica fibrosa. The plate reveals a bone cyst with marked curving of the upper end of the femur.

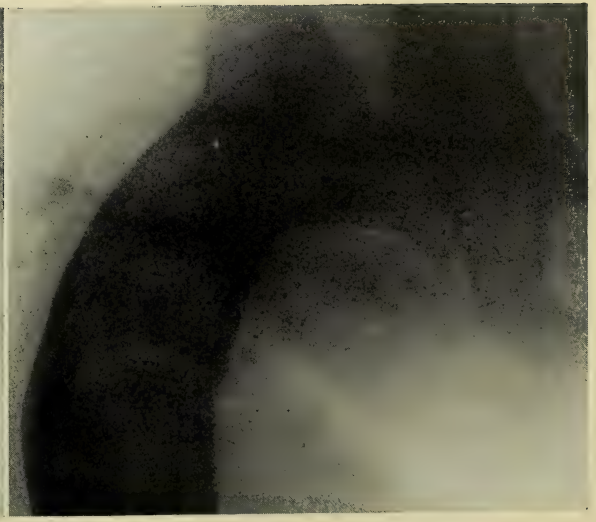


Fig. 22.—Roentgenogram of Case VI after operation. The cavities are almost filled with osseous material.

Fig. 23 Fig. 24

Fig. 23.—Roentgenogram of Case VII showing disease of the fourth lumbar vertebra. A safety pin is used as a marker.

Fig. 24.—Roentgenogram of Case VII showing the Albee bonegraft and correction of the deformity.

Fig. 25 Fig. 26

Fig. 25.—Lateral view of Case VII showing almost complete correction of lordosis.

Fig. 26.—Photograph showing back and operative scar.

Fig. 27.—Roentgenogram of Case VIII showing exostosis of humerus.

Fig. 28.—Roentgenogram of Case VIII showing condition after removal.

sisted for sixty years with recurrent exacerbations, resulting in invalidism during the greater part of the time and a cripple harnessed with hip splints during the remainder. Diagnosed and treated as tubercular hip, and when finally presented for radical cure came with a complete bony fusion of the entire joint, with destruction of head of femur and consequently poor material with which to form a new and serviceable joint; added to this the pain status was so firmly established that it is doubtful even with a perfect anatomical result if it will ever be abolished. Therefore it was decided to do a radical operation and obviate, if possible, long confinement to bed with only temporary benefit under the most favorable conditions, and avoid loss of the essential bony framework necessary in joint reconstruction. On October 9, 1917, arthrodesis was done, exposing the joint through a U-skin incision. On opening the capsule a brownish fluid, with flakes of coagulated fibrin, was found filling the joint. After incision of capsule the head of the femur was readily dislocated, owing to destruction of its round ligament. All synovia were very red and swollen, evidencing the acuteness of the inflammatory process. The acetabulum was deepened and conical in outline. All inflamed synovial surfaces were excised, the acetabulum curetted, reamed out and swabbed with a 5 per cent. tincture of iodin and dried. A flap of aponeurosis and fat was dried and stitched around the acetabular rim and head of femur replaced. Usual steps were followed in completing the operation after Murphy's technic. The patient was placed on a frame with a plastic-bandage fixation and extension applied. Stock vaccine was continued at two-day intervals for two weeks. Highest temperature first day after operation 100° F., 102° for two days, 104° fourth and fifth days, 101° sixth and seventh days, steadily declining to normal during the second week, following this no elevation of temperature. In bed for two months, no pain on movement. On crutches. No limitations of voluntary movement as compared with right hip. Complete restoration (Fig. 4).

Case IV (*bony ankylosis from fracture of knee*).—T. J., colored, aged seventeen years. Struck just above the knee by a piece of timber in September, 1916. Treated for fracture of thigh. Presented at Burwell Infirmary January, 1917, for stiff knee. Unable to walk without crutches. Bony ankylosis of knee at angle of 75 degrees. *X*-rays showed transverse fracture through lower epiphysis of femur, upper fragment displaced, forward impinging against patella, large callus posteriorly extending two and a half inches upward along femoral shaft. Operated January 29, 1917. Following technic of J. B. Murphy the joint was entered through bilateral longitudinal incisions. Patellas were freed from the fractured end of the femur with chisel. A transverse osteotomy of the lower end of femur was done, following the fracture lines, and the fracture reduced. A lateral phosphor-bronze wire suture was introduced to retain the fractured femoral end in apposition. A small flap of fascia lata was thrown across the denudation of the articular surface of patella and held in place by chromic catgut sutures on both sides of patella. Wound closed; buried suture of chromic catgut, skin sutures of silkworm gut. Plaster-of-Paris bandage applied over gauze dressing, extending from crotch to toes. No extension necessary for reason that articular surface of femur and tibia were not injured, and the flap interposed was not subjected to pressure. Uneventful recovery. Good motion in joint. Walks without limp (Figs. 5 and 6).

Case V (*bony ankylosis from fracture dislocation of elbow*). —Colored girl, aged fifteen years. Fell from wagon December, 1916, dislocating and fracturing elbow. Rigid ankylosis of joint almost at right angle. Arm in most useless position. Murphy arthrodesis done March, 1917. Results shown in photograph (Figs. 7 and 8).

Case VI (*osteitis cystica fibrosa*).—Mrs. P. D., married, aged twenty-seven years. Family history negative. Personal history negative, except that at the age of eleven she fell suddenly while walking, striking her right hip. Being

unable to arise from the ground, she was taken home and treated for fracture of the hip; in bed six weeks, with extension applied. Noticed right leg shorter, and as she began to walk it appeared that the shortening was slowly but progressively increasing. From time of injury the hip has never been without discomfort. A feeling of fulness deep in, with occasional sharp pains. Admitted to Vaughan Memorial Hospital December 16, 1916. Fleshy woman with four inches' shortening of right limb, bowing of right thigh. Very slight limitation of motion and no pain in joint. Slight tenderness over upper half of shaft of femur, palpable enlargement of shaft of femur.

X-ray exposure showed cystic degeneration, with consequent bending of upper end of shaft of femur. A case of osteitis cystica fibrosa described by Murphy of metastatic origin. Operation done December 12, 1916. A section of bone was removed with twin saws over all cystic area. Cysts curetted thoroughly. Trabeculæ dividing them were not disturbed. Into these large cyst cavities were placed bone clippings, using segment removed for this purpose. A graft was then removed from the left tibia, including the periosteum and endosteum, and fitted into the hiatus. Several cross-sections of the graft were made with the saw, in order that it could be bent to conform to the shape of the curved femur. Recovery and healing of wound occurred without incident. Subsequent *x*-ray picture shows filling of cystic area with bone. The absence of opening and sense of fulness in leg is evidence of cure of the disease. Her tonsils were removed, and she was referred to a dentist for necessary work of teeth. Discharged from hospital January 14, 1917 (Figs. 9 and 10).

Case VII (*Pott's disease; lordosis*).—T. E. P., white, married, aged twenty-eight years. Boilermaker. Member of large family. History negative, except one brother, who has tuberculosis, but not living in the same house with him since it was contracted. Never ill except attacks of malarial fever in 1907, and frequent attacks of tonsillitis during childhood. No injury except strained arm, caused by fall

from house when ten years old. Present illness began in 1911, with pain in sacrolumbar region. Unable to turn over if lying on back, tired easily and had to rest frequently while at work. Negative report from *x*-rays made at that time. In 1915 he quit his trade of a boilermaker and opened a retail grocery, with less discomfort. Entered Navy Yard at Philadelphia in 1916, but was unable to stand the work, and came to Montgomery to work at his trade, but was forced to give it up. Two months later, September 30, 1917, he entered the Vaughan Memorial Hospital on account of pains in back and limbs. Frequently falling when on feet and inability to walk any distance. Examination showed lordosis. Spine of the fourth lumbar vertebra was displaced forward, forming a decided depressed space over his vertebra. *X*-rays showed displacement and partial destruction of the body of this vertebra. On October 1, 1917, Albee's operation was done, interposing a tibial graft between the split spinous processes of all five lumbar vertebræ and the spinous process of the sacrum. Kept on frame two and a half weeks; in bed two months. Recovery without incident. His back feels strong and he is anxious to resume work (Fig. 7).

Case VIII (*exostosis of humerus*).—W., male, white, aged forty years; single; cigarmaker. Past history negative. Stiff and painful shoulder. Consulted me October 9, 1917, complaining of inability to raise his arm and of pain on any movement of the shoulder. Tender on palpation over the deltoid. Passive movement painful. No ankylosis. Temperature and pulse normal. No history of injury. *X*-ray plate showed exostosis near joint; situated a half-inch from articular surface of upper end of humerus. Operation done October 9, 1917. On chiselling through the base of the growth it was found to be a thin shell of bone filled with a cheesy mass and dipping deep into the shaft of the bone. This was a tubercular infarct. This cavity was thoroughly curetted and filled with a strip of muscle tissue, reflected from the deltoid, and held in place by a catgut suture. *X*-rays show result obtained (Figs. 13 and 14).

DISCUSSION ON THE PAPERS OF DRS. REDER, SHANDS AND DU BOSE.

DR. JOHN E. CANNADAY, Charleston, West Virginia.—I wish to compliment Dr. Reder on his persistence in bringing such a calamitous case to a successful issue. I have not in my experience observed the painful muscular contractions which he has described. In my own practice following operation, bone transplants, etc., on the long bone, I apply a plaster cast, having the knee or elbow, as the case may be, slightly flexed.

The researches of Macewen, Ollier and Barth, also more recently of Barney Brooks, show that both the periosteum and endosteum are of great advantage in promptly securing new vascular connections following the bone-graft operation. Dead bone or foreign bone has no advantages whatever over the metal plate. It acts as a foreign body would. An autogenous bone transplant takes hold and becomes a part of the living tissues.

In a recent case of my own in which a bone transplant was implanted after resection of a tubercular knee in the presence of a mixed infection I was obliged some weeks later to do an amputation, owing to the prolonged suppuration about the joint. To my surprise on dissecting the part I found the graft that I had placed eight weeks before was still alive and had a definite vascular connection by way of its periosteal covering.

I should like to ask Dr. Shands if these splints are inflammable or not. I have always felt that celluloid was highly inflammable because I have known of one or two fatalities resulting from the inflammability of celluloid.

DR. F. G. DU BOSE.—I wish to thank Dr. Reder for his remarks. I think one word of warning ought to be given in reference to snugly fitting plaster. Some of us have seen a few cases of Volkmann's ischemia resulting from snugly fitting bandages early in the treatment of fractures, on account of no provision having been made for swelling, and any man who has seen this ischemia result in cases of fracture treated by other surgeons feels very leary about snugly fitting bandages in any fracture because of the possibility of swelling of the parts after the bandage is applied.

Dr. A. R. Shands, Washington, D. C.—It will burn, but it can be rendered absolutely non-inflammable if you add chloride of lime solution to the acetone; this makes a material similar to asbestos. I have never found this necessary.

Dr. Alexius McGlannan, Baltimore, Maryland.—One point in regard to the treatment of fractures has been emphasized repeatedly, for those cases in which it seems very difficult, if not impossible, to avoid violent muscular contractions. The proper thing to do is to put the limb in a position in which the muscles are the nearest at rest, so that in a fracture of the femur, if the dressing is made with the knee and hip each partially flexed, this will prevent a great deal of damage through the pulling of the muscles. When plaster of Paris is applied to a limb with the joint flexed we do not depend upon circular tension of the cast to maintain the fixation but on the length between the flexed angles, and there is no constriction, and no interference with the free circulation which is so essential to the union of bone, and I believe our results in fracture of the shaft of the femur are due to the fact that we put them up in this position.

Dr. A. R. Shands, Washington, D. C.—I have had an extensive experience in the treatment of fractures of the femur and never have encountered the violent muscular spasms that Dr. Reder refers to.

Dr. Francis Reder.—This was not at all a unique case. It was just an ordinary case. The patient informed me from the beginning that she had a great deal of trouble with muscular spasms, but I did not pay much attention to her statement. It was the first time I ever encountered such violent muscular contractions.

My experience in fractures with the shaft bones has been quite extensive, especially in my service at the City Hospital, where we have from 10 to 20 cases of fracture most of the time. In none of the cases have we encountered such muscular spasms, and surely some of them had good cause for muscular contractions.

CRANIAL DECOMPRESSION FOR HEAD INJURIES ACCOMPANIED BY SIGNS OF INCREASED INTRACRANIAL PRESSURE

By R. L. Payne, Jr., M.D., F.A.C.S.
Norfolk, Va.

Since 1909 I have written several papers on the subject of "Fracture of the Base of the Skull," reporting some of my work and recommending that temporal decompression be always done whenever these cases develop a certain degree of increased intracranial pressure.

From that time up to the present I have been astonished at the indifference many doctors show, especially those doing and seeing little surgery, to this subject of severe head injuries, and I wish to report again my small experience in this work.

In considering the value and advisability of subtemporal decompression in skull and brain injuries the burden of proof would naturally fall on the results obtained with and without operation in a given series of skull fractures. Unfortunately, up to the present time, these statistics have been based on the total number of fracture cases coming under a given surgeon's experience, and I here wish to express the view that such deductions are erroneous and do not permit of truthful conclusions.

I think we will all agree that demonstrable depressed fractures and those showing focal symptoms need some operative treatment, and with this type eliminated there is then left two distinct varieties of head injuries—namely, mild and

severe—in which the classification depends entirely on the surgeon's ability to determine whether there is or is not a marked increase of intracranial pressure. In the mild type the intracranial pressure is slightly raised, soon returns to normal and there is no necessity for an operation; but the question of subtemporal decompression in these cases depends largely on the accurate determination of just what the intracranial pressure is doing. The very fact that this determination can be definitely carried out at once places these mild cases beyond possible operative interference, and therefore should eliminate them from statistics presented, with the view of estimating the value of subtemporal decompression in head injuries.

It is only in the severe types of head injuries with marked increase of pressure that decompression should be considered, and therefore the severe variety alone should be included in records collected for the purpose of determining the value of subtemporal decompression in these cases. On the other hand many of these severe head injuries suffer profound shock and such severe damage to the medulla that death occurs at once or in a few hours. These cases therefore never come within the realm of an operative possibility, and hence should not be considered in our statistics. I think we might say approximately that a person with a severe head injury living eight hours after injury should be considered an operative possibility, and studied carefully with the view of determining whether a decompression should be done. Many cases, however, recover from the initial shock within the first one or two hours and in these instances investigation of the various clinical signs of increased pressure should be started at once. When the increase in intracranial pressure rises definitely high, one can never tell how soon the fatal stage of medullary collapse may occur, therefore if operation is indicated it should be done as early as possible.

The greatest mistake now made in the treatment of these cases is that of waiting too long when operation is indicated.

Some of the operative deaths have undoubtedly been due to interference before recovery from the initial shock, but the greatest error made in these cases and the hardest to escape from is that of delay when once the intracranial pressure has reached a high degree. This brings us to the consideration of what are the indications for subtemporal decompression in these cases.

If a patient sustaining a head injury does not die within the first few hours there is first recovery from the initial shock, the blood-pressure gradually rises to about normal and then the signs of increased intracranial pressure may be studied for the first time. These signs are to be found in the pulse-rate, the eye-grounds, the spinal pressure and the systemic blood-pressure. The pulse-rate and general blood-pressure should be recorded every thirty minutes, the eye-grounds examined every hour and the spinal pressure estimated one or more times according to the indications for or against this procedure.

If there is an extremely high rapid rise in intracranial pressure there will quickly develop medullary compression, with edema, and in this state we will have a fast, increasing pulse-rate, a fall in blood-pressure, negative eye-grounds and a low spinal pressure. It is needless to say that this is the fatal state, and any operation undertaken in this stage would only hasten the end. If the rise in intracranial pressure is slow but gradually increasing there will occur a slowing of the pulse, engorgement of the retinal veins and a rise in both the systemic blood-pressure and spinal pressure.

Some surgeons do not attach much importance to the question of blood-pressure, and in this connection it is to be recorded that we do not get an increase in blood-pressure until the rise in intracranial pressure equals that of the general circulation. Irritation or stimulation of the vasomotor centers then occurs, the general blood-pressure rises a step of from 5 to 15 mm., and is simply Nature's method of overcoming medullary anemia. There is never an increased

blood-pressure early when associated with only a mild increase of intracranial pressure. I have found that blood-pressure readings are often of material value, and once this pressure has risen high and begins to fall an operation in my experience has always been futile.

When the intracranial pressure attains sufficient height the return flow in the retinal veins is obstructed, with a resulting dilatation, and if the condition progresses there will occur first an obstruction of the nasal half of the eye-grounds and later a total blurring will develop. This sign will often be found in only one eye, as several of my cases have shown.

In considering the four cardinal signs of increased intracranial pressure I feel that the change in pulse-rate is not only the most readily observed but the most dependable symptom in our present knowledge. This is probably due to the fact that of the three vital centers in the medulla the vagus is not only the most stable but responds more rapidly to stimulation. When a pulse of 80 or more gradually falls to 60 we should strongly consider a relief of the intracranial pressure. A greater drop to 55 or 50, coupled with retinal edema and a rise of the blood and spinal pressure, is positive indication for an immediate decompression.

The value of spinal pressure estimations in these acute cases is sometimes valuable but oftentimes dangerous. It should never be done when there is a high blood-pressure or during the stage of marked increase in intracranial pressure. Several deaths have been reported following this mistake. This procedure is probably of more value in subacute and chronic cases, and should only be utilized early in the acute cases before there is a decided rise in intracranial pressure.

It is a mistake to wait in these cases for a development of all the signs of increased pressure before operation is undertaken. Experience after all is the best guide, and watching some of these cases will teach a surgeon to decide upon operation when only two of the cardinal signs are present. For instance, a pulse of 50 coupled with partial optic edema,

or a pulse of 50 coupled with a rise in spinal pressure to 20 mm. on a mercurial manometer, or a slow pulse, a rising blood-pressure of two steps coupled with either optic edema or increased spinal pressure is entirely sufficient to warrant operation.

In grave cases with very high intracranial pressure unrelieved by decompression there will occur, sooner or later, an exhaustion of the vasomotor and vagus centers. This is indicated by a rapidly rising pulse-rate and a falling blood-pressure, and invariably means medullary edema and approaching medullary collapse. These symptoms following a previously slow pulse and high blood-pressure in such a case are therefore positive contra-indications for operation. I have never seen a case of acute high intracranial pressure with a pulse of 45 to 50 recover by operation if the case was left unoperated until the pulse on the secondary rise had reached 95. If there is any hesitation whether operation should be done in a case showing the cardinal signs of a high increase in intracranial pressure, this doubt can be at once dispelled if the pulse-rate suddenly increased fifteen to twenty beats.

Text-books quote the mortality of basal fractures from 50 per cent. to 90 per cent. The most reliable data in print today are from Dr. William Sharpe. Carefully analyzing his reports (*Op. Therapeusis*, Johnson, vol. ii, p. 656, and *Surg., Gynec. and Obst.*, January, 1917, p. 30) would permit one to arrive at the following deductions:

From first report to June 1, 1914:

Fifty-seven cases possible for operation but not necessarily indicated.

Twenty-one cases not operated: 3 recovered, or 14 per cent.; 18 died, or 85 per cent.

Thirty-six cases operated: 27 recovered, or 75 per cent.; 9 died, or 25 per cent.

From second report to June 1, 1916:

Eighty-six cases possible for operation:

Eleven cases not operated—all died—100 per cent.

Seventy-five cases operated: 54 recovered, or 72 per cent.; 21 died, or 28 per cent.

In arriving at these deductions from Dr. Sharpe's cases we have eliminated, as previously mentioned in this paper, those cases having only a slight temporary rise in intracranial pressure and the moribund head injuries dying a few hours after admission. Certainly these two types would never come within the realm of an operative possibility and therefore should be excluded from statistics bearing on the value of the operative procedure. For the same reason I herewith report my own experience of 29 head injuries accompanied by signs of high intracranial pressure in which the operation of subtemporal decompression was done on every case.

Twenty-two, or 75.86 per cent., of these cases recovered while 7, or 24.14 per cent., died. Some of the deaths in my cases, occurring in my earlier experience, were undoubtedly due to delay in operating until medullary edema had begun. In my last 7 successive cases chosen for operation there has been only 1 death following decompression.

In considering statistics there is further chance for error as follows: (1) because the patient died without operation, that therefore he would have lived if operation had been performed; (2) we say a series of cases might be possible for operation; this is tentative, depending on their condition, and certainly does not mean that operation is indicated in all of them; (3) I do not think that any surgeon can assume, just because operation resulted in recovery, that therefore the patient would have died without operation.

Some of these high-pressure cases occasionally recover without operation, but practically all of them have to reckon afterward with post-traumatic brain and general nervous symptoms which, it is reasonable to believe, would have been forestalled by a timely unilateral or bilateral decompression.

In view of the operative recoveries reported and the absence of post-traumatic symptoms following a timely decompression I again take the position that our results will be much better in these cases if, with the proper indications present, we help relieve the threatened danger to the medulla and rest of the brain by a cranial decompression.

DISCUSSION

Dr. Alexius McGlannan, Baltimore, Maryland.—I have been through very much the same experience as Dr. Payne, and I wish to congratulate him on his excellent results and to thank him for his splendid paper, presenting, as he does, valuable facts and conclusions.

But there is one thing I should like to add, and I do so with a proper degree of respect for his opinion, and that is, a study of the respiratory rhythm in connection with the blood-pressure in making up one's mind for or against immediate operation in one of these patients suffering from high intracranial pressure. We have come to believe that alteration in the rhythm of the respiration is an indication for opening the head. When a patient, who has a slow pulse, a high blood-pressure and stertorous respiration, begins to have respiratory rhythm approaching the Cheyne-Stokes type, that is, begins to take a deep breath, and occasionally a shallow one, it is an indication to open that individual's head without waiting for any further change in pulse or respiration. I should like to ask Dr. Payne if he has noticed this phenomenon and whether he has had any experience regarding this point, as I think it is one of value in determining the type for operation in these head injuries.

Dr. W. R. Jackson, Mobile, Alabama.—I should like to ask Dr. Payne a question with reference to the technic, namely, what is the extent of his temporal cerebral decompression, how much of the area of the temporal region does he include in his excision or decompression operation, and does he always open the dura? If so, does he suture it back again?

Dr. Francis Reder, St. Louis, Missouri.—I should like to ask Dr. Payne whether he makes a double decompression, and if so, what is his reason for so doing?

In head injuries, including gunshot injuries, we have had recourse to, unless the patient was *in extremis*, enlarging the point of entrance to the size of a silver dollar, thereby hoping to anticipate any edema, and in that way avoid to some extent compression of the brain.

The doctor's results have been extraordinary in this class of injuries. I cannot say that we have such results in the City Hospital where we get many of such cases. We subject about one-half of the cases to decompression, the other half we do not operate on. Occasionally we are very agreeably surprised to have a patient recover when an operation had been contemplated but not carried out, and again we feel that in some cases life might have been saved by a decompression operation. It is a very difficult matter to judge when such a patient should be subjected to operation. I appreciate Dr. Payne's paper very much.

Dr. James E. Thompson, Galveston, Texas.—I should thank Dr. Payne when he closes the discussion to make clear to me the following points which I failed to understand when he was reading his paper, namely: (1) the indications for and against spinal puncture, and (2) the indications for and against the decompression operation as shown by the pulse, respiration and blood-pressure.

As regards spinal puncture, I have been almost in the habit of using it as a routine procedure in serious head injuries. In one case of head injury which came under my care three weeks after the accident, I punctured almost every day for the first week. At first the fluid was under great pressure, but at the later punctures very little fluid flowed through the needle. The patient was unconscious and at times very violent. Eventually I performed a temporal decompression on the left side. I found after opening the dura mater that the brain was shrunken and I could pass my forefinger between the dura and cortex. The patient eventually made a complete recovery after almost two months of unconsciousness.

Dr. Payne (closing).—In reply to Dr. McGlannan's remarks relative to the respiratory center, this has been reported in every

text-book that we pick up and in most of the older monographs. We do not pay any attention to it for the reason that when the respiratory center begins to show a change in its rate and character it means that the medulla is beginning to get up a pretty good degree of compression, and this is the Cheyne-Stokes respiration. It is the advanced stage of medullary compression in which the irritation first increases the pressure, stimulates the vasomotor mechanism, which acting as an irritant increases the vasomotor contraction in the arterioles. That in turn increases the flow of blood to the medulla, and this renewed supply of blood to the medulla is what makes the respirations pick up temporarily. That is really Cheyne-Stokes breathing; therefore, no dependence was put upon the respiratory action as an indication for operation, and decompression should be undertaken long before the respiratory centers begin to fail.

With reference to Dr. Jackson's question about the technic of decompression, for several years I made the ordinary horseshoe flap incision. For about a year now I have made use of a vertical incision, starting at the temporal region and going through the temporal muscle to the zygoma, the muscle is retracted to either side and you can go into the skull by whatever method you prefer. There is no better instrument for opening the skull than a Carr drill. It beats the Hudson drill. I usually make four small punctures in the form of a double triangle and follow that with a motor trephine spiral drill. You should open the dura and any decompression that does not include incision of the dura is an incomplete operation. If you open the dura and then get a subdural clot to drain with a small piece of rubber packing you cannot use the osteoplastic flap. You do not have continuous decompression. The temporal muscle covers the defect and that is carried out in the vertical incision.

Dr. Jackson.—You do not replace the bone?

Dr. Payne.—That is taken out and left out. In reply to Dr. Thompson, spinal puncture is more or less new in this business. None of us know a great deal about it, and for many years we depended upon the mercurial manometer. A good many cases have been reported in which spinal puncture was undertaken for diagnostic purposes and sudden death occurred from sucking the medulla into the foramen magnum. Spinal puncture therefore is attended by more or less danger. The general blood-pressure does not rise until the intracranial pressure rises

to the point where it is equal to the general blood-pressure; then that acts as an irritant to the vasomotor centers of the medulla and you get a corresponding rise in general blood-pressure of from 5 to 15, possibly 20, millimeters of mercury. You only get that when you watch the blood-pressure from the start, and it is still low. That is the reason so few clinicians pay any general attention to blood-pressure as a valuable sign in these cases. If that is the case, as stated, a high general blood-pressure would cause a very high intracranial pressure correspondingly. If you have a very high systolic blood-pressure, that possibly would serve as a contra-indication for the special estimation of the pressure, the withdrawal of spinal fluid, because it would result possibly in the foramen being plugged up by the medulla.

As to double decompression I hardly know how to answer that question. In my paper I said that experience is the best teacher, and a man after he has seen a few of these cases can certainly decide when to operate from the cardinal signs rather than wait for others to develop. The same rule applies when it comes to double decompression in cases of very high blood-pressure in which the pulsation is absolutely obliterated and in which we must tap the ventricle to get relief. Such patients ought to have bilateral decompression, and that conclusion we have reached only recently. I have done bilateral decompression three times. In one of these cases we did it as indicated. In the other two instances we did a unilateral decompression in which the patient's symptoms did not improve. The pulse did not rise; the vagus remained low and we deemed it wise to operate and do a decompression on the other side. One of these patients lived and the other one died.

INJURIES OF THE CERVICAL SPINE

By Joseph Ransohoff, M.D., F.A.C.S., F.R.C.S. (Eng.)
Cincinnati, Ohio

A broken neck has from time immemorial been potential of a speedy death, and if the fracture is of the right kind, recovery is impossible. As if the break in the cervical spine were insufficient there quite often occurs with it a fracture of the base of the skull to make the end doubly sure.

Within two years we have had in the Cincinnati General Hospital no less than 3 cases of fracture of the cervical spine, with fracture of the base of the skull. In one of these there was also a fracture of the fifth lumbar vertebra. In each of these cases there was a great deal of shock, a condition which curiously enough is not ordinarily associated with cord injury. In one of these cases the temperature rose to 110° four hours before death and forty-eight hours after the receipt of the injury.

Even in very high fractures shock is often absent. In the case of a man who died on the fourth day after injury there was a found a fracture with dislocation of the axis. Although this man lived nearly four days there was no shock, and notwithstanding the extensive derangement in the atlas there could have been only a partial cord disintegration.

The *x*-ray findings of fracture dislocations of the cervical spine, with extensive disorganization of the cord, are often disappointing, since they fail to reveal a degree of derangement of the column commensurate with the cord injury.

For example, in the case of Sheridan, A–4115 (Fig. 1), where death resulted after a subtotal paraplegia, the roentgenogram must be carefully studied before the break in the anterior lower margin of the fifth cervical body can be seen. There is no displacement. There was no weakness in the upper arm and no loss of sensation. There was marked weakness of the flexors and extensors of fingers and hand. The line of demarcation began at the third intercostal space, below which there was complete anesthesia. Below this line there was flaccid paralysis and loss of deep reflexes. There was extreme arteriosclerosis. Sloughs formed on the buttocks and heels in a few days, but the patient lived seventeen days.

The case of B–7671, to be shown later, further illustrates this point. A fine abnormal line between the transverse processes indicates where the dislocation had been. Except for some control over the deltoids there is a complete flaccid paraplegia. Exitus occurred sixty hours after injury was sustained.

This discrepancy between the roentgenographic findings and the cord changes are doubtless due to the fact that after the accident, either spontaneously or by the handling of the patient, a reposition of the parts takes place to something like their normal relations. Particularly is this true of the cases in which luxation plays a great role. While Chipault opposes this view of spontaneous replacement, Thoburn believes that in the cervical region particularly, reposition is twice as common as is the permanent displacement. Our study of the roentgenographic findings, as we have seen, supports the contention of the English surgeon, and brings into the limelight the futility of interfering for deformities of the column alone, since the spontaneous relief of the latter, or that following manipulation, fails in so many instances favorably to influence the damaged medulla.

There were altogether 14 subcutaneous injuries to the cervical spine, of which 6 died. The complications in the fatal cases were fractures of the base of the skull, as already

alluded to, or other internal injuries; 8 of the cases recovered, and of these 6 presented neither cord nor nerve-root symptoms of any kind; 2 of the cases presented cord or cord and root symptoms. In addition to these there were two cord lesions from penetrating wounds, one of which died.

In most of our fatal cases the injury was to one of the lower cervical vertebræ, the place of election where the more movable portion joins the more fixed segment at its junction with the upper dorsal. One exception was in a case of fracture of the third vertebra. The other was a fracture dislocation which was admitted to the neurologic service, while in a post-epileptic mania. In an attempt to escape from the police he jumped from a window and sustained his injury. A satisfactory neurological examination could not be made, since the patient was deeply comatose. The pupillary reaction was sluggish, the left pupil being the larger. Both arms and hands are moved without intent, and both legs, although paralyzed, respond somewhat to plantar irritation (Townsend, Fig. 2). The patient died within seventy-two hours, in deep coma.

Although the roentgenogram failed to reveal any injury to the skull it is certain that death was due to a cerebral lesion. The roentgenogram shows a fracture of the odontoid process, with extensive comminution of the atlas.

In none of the fatal cases was an operation performed. In withholding from surgical interference, my colleagues and I evidently incline to heed the dictum of Horsley, to wit: "If the lesion is acute and in the cervical region, then certainly wait. As regards the dorsal region, better to wait a little. As regards the lumbar region, I do not think you want to wait to operate."

In a report from the Massachusetts General Hospital, by Hartwell, from 133 fractures of the spine, 40 patients were subjected to operation, 38 to laminectomy and 2 to an attempt to remove the deformity by manipulation. Taken as a whole the result of expectant treatment in these cases

equalled those obtained by the operative treatment, and the results of laminectomy do not justify an argument in favor of operation but rather serve as a warning against radical surgical treatment. Indeed, there are some cases in which an operation seems positively indicated, and in which, if performed, would be held responsible for recovery if it took place. But such cases seem quite as often to recover without an operation.

The following case observed in private practice twenty-three years ago illustrates a complete recovery, after more than a year, from what appeared to be a severe transverse lesion of the cord in the fifth cervical segment:

L. J., aged twenty-seven years, on November 24, 1895, while diving in shallow water, struck his head on the concrete bottom of a pool. He remembers floating to the top of the water, but could not lift his head out of the water. He did not lose consciousness and felt that he might drown before anyone discovered him. Assistance came, however. It was after he was brought home that he felt that he was completely paralyzed from the shoulder down and that the paralysis was not complete from the start. An examination made by Dr. Maury showed a fracture of the fifth and sixth cervical vertebræ. He was treated by Dr. Maury with a plaster cast and head extension. When this man was seen by me nine months after the injury the condition was practically unchanged. After the year the paralysis gradually improved, and now, twenty-three years after the accident, there is slight weakness of the arms in lifting and some of the muscles of the shoulders have slightly atrophied. The roentgenograms, which were recently taken by Dr. W. S. Lawrence, of Memphis (Figs. 3 and 4), show that there is an almost complete coalescence of the fourth, fifth and sixth vertebræ.

In this connection the case of F. M., aged twenty years, is also interesting. Unfortunately the roentgenograms cannot be found. On the day before admission the patient fell down some steps and was rendered unconscious. For a

short time after gaining consciousness she was irrational and confused. There was localized tenderness over the fifth and sixth cervical spines, and crepitus could be easily obtained. There was paralysis of both arms below the shoulder with almost complete paralysis of the triceps and extensor muscles of forearms and fingers on both sides. A great deal of pain is complained of in the arms, but not well localized. Touch sensation is entirely absent over the dorsum of both hands and the extensor surfaces of the forearms, and is much impaired over the left arm to the shoulder girdle. Pain sensation over the dorsum of the hand and the extensor surfaces of the forearms is entirely abolished. Pain sense is also diminished over the flexor surfaces. There is paresis of both legs, more marked in the left. The dorsal extensors of the foot seem chiefly affected. There is urinary and fecal incontinence.

The neurological diagnosis read: "Destruction of rootlets, both anterior and posterior, coming chiefly from the sixth cervical segment, resulting in almost complete paralysis of the triceps, extensors of the hand and fingers, the major pectorals on the right and left side. Also a zone of sensory destruction covering the back of the hand and forearm."

From the involvement of the lower extremities and the loss of functions of vesical and anal sphincters an incomplete cord lesion was surely present. When this patient was discharged from the hospital within two months she had entirely recovered, except for a paralysis of the extensor muscles of the fourth and fifth fingers of the left hand.

Another case, with even more serious cord lesion, that quickly recovered without operation, was that of Spencer Lynn (A–4146, aged fifty-seven years, Fig. 5). Falling down stairs he sustained a fracture of the spinous processes of the third, fourth and sixth cervical vertebræ. Pain sensation in the upper extremities was normal; absent in the lower. There was muscular paralysis of the lower extremities to the hips; patellar reflexes were much exaggerated; ankle-clonus;

plantar reflexes normal. There was no priapism, incontinence of urine or feces.

The lesion here was evidently not a complete transverse one. This patient left the house two months after admission. walking without assistance and with complete control of his sphincters.

In these cases, which recovered without operation, the recovery would have been attributed to the operation had one been performed.

The mortality of our cervical spine injuries is less than 50 per cent. This is in marked contrast with those recorded only a few years ago. Chipault had 22 cases, with 21 deaths and 1 improvement; Bergman, while at St. Petersburg, had 16 cases, with 16 deaths; Guy's Hospital had 36 cases, with 36 deaths; Frank Hamilton had 18 cases with 18 deaths. The cause of this discrepancy is of course the graphic manner in which injuries to the spine are now displayed, white on black. Our immediate forebears in surgery based the diagnosis of spinal lesions almost wholly on the narrowing of the spinal canal which they produced and the damage to the cord consequent thereon. The localized pressure tenderness, muscular rigidity, restricted movement and irregular alignment of the spinous process were of necessity only potential factors toward diagnostic probabilities. Fractures of the spinous processes were thus recognized, but these are, as a rule, not limited to the spines but involve the bodies as well. The roentgenograms of cases A–2614 (Lang, Fig. 6) and B–2544 (Hampton, Fig. 7) show this admirably. As to the neurological findings, both cases were negative. They did not make the diagnosis certain. As we have seen, the outcome in the class of cases formerly regarded as fractures of the spine is no less or little less fatal today than it always has been. It is self-evident that we now recognize, with greater ease, injuries to the vertebral column.

The taking of plates through the open mouth, as first advocated by Griffiths, is a valuable addition to the x-ray

study of the cervical spine. Fig. 8 shows a bullet located by a roentgenogram taken through the mouth. Although it seems to be lodged against the spine, it is behind the spine and has not touched it. The track of the bullet can be easily followed from the point of entrance in the parotid region to its final lodgment behind the spine.

The frequency with which the cervical spine is injured without cord involvement is illustrated by the number of cases recorded in less than two years in the Cincinnati General Hospital. The experiences in this hospital is of course not unique. Hartwell, during six months' service in the male surgical division of the out-patient department of the Massachusetts General Hospital, reports 10 patients who sought relief for pain in the back and who were found to have a fracture of the spine, and 1 patient with spinal fracture who was treated in the orthopedic division. Not one of the cases had at any time had any motor or sensory paralysis or sphincteric disturbance. Of the series, 8 cases were compression fractures, 1 was a fracture dislocation and 2 were fractures of the transverse processes of the lumbar vertebræ.

Curiously enough the fatal cases in cervical fractures are mostly in the lower four vertebræ and are compression fractures from indirect trauma. We saw no cases of tear of the intervertebral disks, which occurs in less than 1 per cent. of the cases.

Only 1 case could be positively diagnosticated as a direct fracture (A–7521, Weaver, Fig. 9), and this was brought about by a blow with a heavy wooden club. The fracture was through the lamina of the second cervical vertebra, with considerable forward displacement of the body. The lateral movements were very much limited and caused a great deal of pain, and although there was considerable dysphagia, there were no cord symptoms whatever. When one considers the damage done to the vertebra and the amount of displacement and narrowing of the canal it seems

a marvel that the cord escaped fatal damage. The same degree of overlapping from trauma in the lower cervical vertebræ would almost certainly have produced fatal infringement on the lumen of the canal and crushed the cord.

In one other case there was a suspicion of direct injury. The patient was struck by a wagon, but there was nothing to indicate where the injury was inflicted (B–7671, Surrat, Fig. 10), except a slight abrasion over the coccyx. Curiously enough this was the only case of the entire series of a pure dislocation without fracture. The body of the fifth vertebra on the stereoscopic plate appears dislocated on the sixth, but if the pictures were presented without any accompanying history it would be improbable, in my judgment, that anyone would make the diagnosis of dislocation. A white line on one side indicates a diastasis of the articular processes. Nevertheless in this case the Thoburn syndrome was complete and priapism was present. The patient died from complete transverse lesion seventy-two hours after admission. It may be observed that priapism was present in all but one of our fatal cases. It is almost always present when there is a complete cord disorganization, and is therefore a most ominous sign.

In all of our cases, with one exception, the neurological evidences of complete cord injury were synchronous with the injury itself. This is a rule to which there are very few exceptions. Thus of 67 patients from the Massachusetts General Hospital who had signs of cord lesions, in 66 the onset of paralysis was immediate, and in only 1 was it gradual. In this connection the case of L. Johnson (B–8641, Fig. 11), of our series, is of interest: This was a colored man, who received a stab wound of the neck on a level with the fifth cervical vertebra. He walked into the hospital from the ambulance. A hematoma the size of an egg, containing a clot, was cleaned out in the receiving ward and the wound sutured. When the patient was asked to get up from the operating table after the sutures were placed it was found that he could

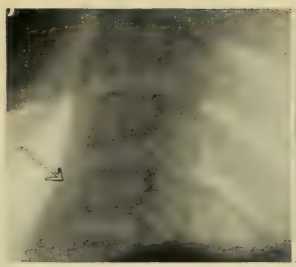


Fig. 1.—(A–4115.)

Fig. 2.—(Townsend.)

Fig. 3.—(L. J.)

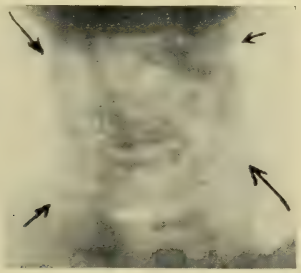


Fig. 4.—(L. J.)

Fig. 5.—(A–4146.)

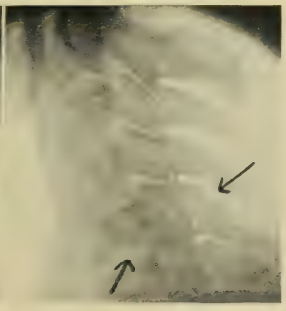


Fig. 6.—(A–2614.)

Fig. 7.—(B–2544.)

Fig. 7.—(B–2544.)

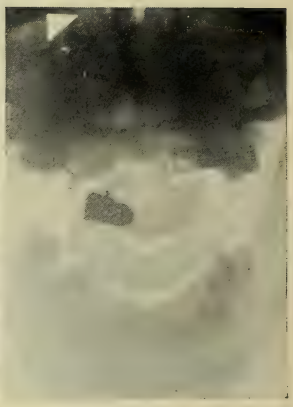


Fig. 8.

Fig. 9.—(A–7521.)

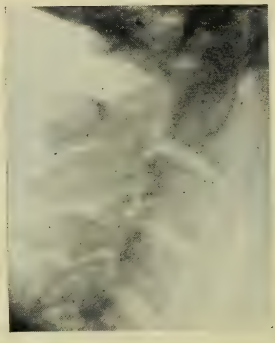


Fig. 10.—(B–7671.)

Fig. 10.—(B–7671.)

Fig. 11.—(B–8641.)

Fig. 11.—(B–8641.)

Fig. 12.—(B–7936.)

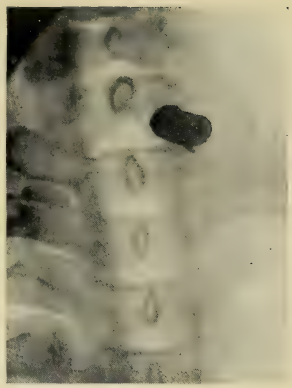


Fig. 13.—(B–6873.)

Fig. 14.—Dislocation of fourth cervical vertebra.

not get up, and on examination a complete paraplegia from the shoulders down was discovered. He was unable to use either leg. The knee-jerks were normal. Evidently the paralysis was the result of a hemorrhage, and for this reason a laminectomy was determined upon. The roentgenogram showed a fracture of the spinous process of the fifth and one of the body of the sixth without displacement. It seems remarkable that the knife-blade could have been withdrawn intact and that the cord was not cut. As happens occasionally in laminectomy in the cervical region that respiration was interfered with, and notwithstanding the establishment of artificial respiration the patient died on the operating table before the membranes could be exposed. An autopsy could not be obtained.

In the treatment of fractures, dislocations and fracture dislocations of the cervical spine we have not only refrained from open operation but likewise from manipulation of a more or less violent nature. In a recent article Stern speaks of 3 cases in which osteopaths and chiropractors have fractured the spine by their violence. We have managed in these cases to get along with suspension and the application of plaster casts of the chest, with head attachments. The end-result even if a reduction of the fracture is not accomplished has been eminently satisfactory in all of these cases so far as we have been enabled to follow them. In the cervical spine, limitation of motion in any one joint, by reason of fracture or dislocation, is easily made negligible by other joints functionating in its place. These patients have thus far not suffered such inconveniences in any instance as to necessitate operative interference for its relief. That does not mean that cord or nerve root symptoms may not develop at a later period. The "sufficient unto the day" adage may be applicable here.

In this connection I am of course conscious of the very excellent work of Walton, emphasized by Mixter and Osgood, in the method of reducing unilateral luxations in the cervical

region. Two very interesting cases of dislocation reduced by manipulation are reported by Major Speed in the December (1917) number of the *Annals of Surgery*. In the pure dislocations there can be no question but that an effort should be made at reduction by the Walton method, but in our series there has not been one that has not been quickly fatal from cord lesion. Since all the non-fatal cases were fracture cases primarily the slower method has been followed. The case of B–7936 (Nevin, Fig. 12), without any cord or nerve symptoms, seems to be one of pure dislocation backward of the seventh cervical vertebra, but a careful study of the plate shows a slight fracture of the body. From a medico-legal view-point these minor injuries of the cervical spine, to a less extent of course than those of the lumbar region, are of major importance. No post-traumatic pain anywhere in the back should stamp the complainant as a malingerer until repeated examinations with the *x*-ray have proved negative.

The most interesting of the injuries in the cervical region was a shot wound of the neck. The patient (B–6873, La Force, Fig. 13), a strong colored man, aged forty-three years, was admitted to the hospital on September 21, 1917. On the right side of the neck, on a level with the cricoid cartilage, was the wound of entrance. The hemorrhage from it was very slight. The patient complained chiefly of pain in the right arm and tingling and pricking sensation. The patient was able to move both arms and legs.

Neurological examination by Dr. Hoppe: The left eye is evidently more prominent than the right. The palpebral fissure is wider on the left side. The right pupil is very much smaller than the left and reacts slightly to light. There is no loss of sensation in the distribution of the fifth nerve. Sensation in the leg is normal There is no zone of hyperesthesia. The muscle power of the right arm is diminished so far as the grasp of the hand in concerned. The motor of the triceps and biceps is normal. The deep reflexes in the

upper extremities are slightly diminished on the right side as compared with the left. The sensations of the right arm in all qualities are normal. The sensation of the trunk is normal. The hypogastric reflex is absent on the right side but present on the left. The cremasteric reflex is absent on the right side; normal on the left. The motor power of the right leg diminished as compared with the left. The patellar reflex on the right side is slightly diminished as compared with left and rapidly exhausted. There are no plantar reflexes on the right side; normal on the left. There is no disturbance of sensation in either leg.

Stereoscopic *x*-ray plates showed the presence of a bullet which had apparently lodged against the interspace between the seventh cervical and first dorsal vertebræ, just to the right of the midline.

As the patient failed to improve after nearly three weeks an operation was determined upon. The dissection followed a triangular trapdoor incision along the clavicle and the median line. The sternocleidomastoid was divided and the scalene muscle with the phrenic nerve was thoroughly exposed. The carotid sheath was drawn to one side and the sympathetic exposed and found intact. It was hoped by the exposure of the lateral processes and bodies of the lower cervical vertebræ that the bullet might be reached from above. Unfortunately this was not accomplished and the bullet still is where it originally lodged. The very large wound healed without any wound complication. Whether or not, probably not, the operation had anything to do with the subsequent history it is difficult to state, but at any rate this patient, almost from the time of the operation, began to improve, so that when he left the hospital on November 8, five weeks after the operation, the pupils were of equal size, there was a slight difference still in the palpebral fissures and the muscular power in both arm and leg had been entirely restored.

The case is interesting from a number of view-points.

This bullet did not penetrate the canal, produced no recognizable fracture and still caused by contusion a lesion in the cord, nicely limited to the pyramidal tract and the gray matter underneath, distinctly recognizable by characteristic symptoms. With the cervical sympathetic cord intact there must have been a contusion of the gray matter of the ciliospinal center to account for the enophthalmos, the contracted pupil and the slight palpebral ptosis. This syndrome is characteristic of injuries of the sympathetic.

It is interesting here to note that the first shot wound of the cervical sympathetic in this country was described by Mitchell, Moorehouse and Keen. In this instance there was, in addition to the eye and lid symptoms, hyperemia of the face on exertion, with pain in the eye and light flashes. As the latter symptoms were absent in our case, and as the sympathetic cord was intact, it seems to corroborate Kraus's contention that lesions of the cord are liable to be followed by the vasomotor symptoms named, and that in lesions of the spinal cord proper the symptoms are limited to the pupil and the eyelids. Those interested in the bibliography of this subject will find a complete resumé in *Die Neurologie des Auges* by Wilbrand and Saenger.

Our judgment in delaying operation in this case was sustained by the most extensive report on shot wounds of the spine during the present war in a communication from von Eiselsberg, who based his view on an experience of 35 cases. He recommended, in contradistinction of course to all brain injuries, that it is best to wait four or five weeks until the condition has become stationary before laminectomy is attempted.

Since the above article was put into type a case of forward dislocation of the fourth cervical vertebra was admitted to the second surgical service of the Cincinnati General Hospital. There were motor-root symptoms, involving both upper extremities, but no paraplegia. The radiogram (Fig. 14) shows no evidence of a gross fracture, although by the tearing

off of the anterior common ligament small spiculæ of bone were evidently wrenched from the vertebral body. This condition I imagine is not very uncommon, although it has been demonstrated in very few, if any, radiograms.

DISCUSSION

Dr. James E. Moore, Minneapolis, Minnesota.—I have been greatly interested in this splendid resumé from one of our older surgeons on spinal injuries. I have had my share of experience in injuries of the spine and I am quite pessimistic as to the amount of good we can accomplish. I shall not go into details about that, but my object in rising is to relate one of the most thrilling experiences I have ever had in my whole professional career of forty-five years.

Dr. Law, who is a member of this Association, called me one day in consultation to see a young man, aged between fifteen and twenty years, who had been caught in an elevator, with his chin thrown up as he struck the floor above. It was not a case for refinement of diagnosis. The boy seemed about to die. A diagnosis could be made from the history of the accident and upon deformity. His chin was shoved forward and he had a decided lordosis. We could feel a body projecting forward by putting his finger in the mouth. He became blue. We secured permission to undertake to reduce the dislocation, telling the parents that the boy might die at any time. He was placed upon the table. We took hold of his extremities. Dr. Law took hold of his head as best he could, using various bandages about it to help him and made traction while I crowded my hand into his mouth, making pressure upon the body of the dislocated vertebra so tightly that I dislocated the jaw, but after a little manipulation the dislocation was reduced. This occurred a number of years ago, and the boy is alive and well today.

Dr. James E. Thompson, Galveston, Texas.—I do not feel that this paper of Dr. Ransohoff's ought to go by without some remarks. I have been very much interested in this subject, and in reading the literature on it for a number of years I have been impressed by the fact that from time

to time some papers appear which advise indiscriminate operation in a very disquieting manner. I am very glad that Dr. Ransohoff has brought this subject before us, and I desire to call attention to the fact that by means of the *x*-rays we can now diagnose and localize a large number of injuries of the spine and fractures through the spinous processes which are not necessarily attended by any mischief to the cord itself. These facts become apparent to us as we get more familiar with *x*-ray pictures. Such injuries, of course, are of medicolegal importance, but a large number of them are not of practical importance, so far as the future of the patient is concerned, because most of the cases recover completely.

The injuries that concern us most are those associated with injuries of the cord as well as injury of the vertebræ. I was particularly struck with the outlook given by Dr. Ransohoff in these cases. Thorburn, in discussing operative procedures on fracture of the vertebrae associated with spinal injuries, pointed out very conclusively before the days of *x*-ray pictures that there was seldom marked deformity to be seen in these fractures, and that when the patient was placed in the recumbent posture the vertebræ dropped into place and all displacement disappeared. If we follow these cases to the postmortem room we can verify this statement any time.

I have in our museum two specimens of fracture of the cervical vertebræ. One was a fracture dislocation between the second and third cervical and the other a fracture dislocation between the third and fourth vertebræ. In both of these the cord was crushed and death was instantaneous. At the postmortem examination scarcely any deformity was present. These specimens were placed in preservatives and the contour of the vertebræ is just exactly as it was during life. One can see the tear in the intervertebral disks and the bodies of the vertebræ distinctly, but no deformity.

As to hemorrhage, hemorrhages are mentioned in the literature as having occurred in the interior of the spinal canal, and are mostly found in cases of contusion and laceration of the cord itself. Even when the cord is squashed like a crushed banana, hemorrhages are very small. We have only to consider two factors: the first, deformity; the second, massive hemorrhage. If we do not have either of these, what can we hope to do by means of operation? We ought to put ourselves on record as opposing very seriously operation on these cases. There is no indication whatever for it.

Dr. Ransohoff brought out the point that the cord may be injured without the canal being encroached upon. That we know occurs quite often. I remember one case particularly: A colored man was shot through the bodies of the upper thoracic vertebræ, who had total immediate paralysis from the time of the accident. He died forty-eight hours afterward. At the postmortem examination the pathologist opened up the spinal canal and found nothing. He could not find the track of the bullet. There was no hemorrhage in the canal either outside or inside the dura mater. On opening the dura mater the cord was seen to be contused over a slight area and microscopically degenerative changes were found. The bullet had passed through the vertebral body opposite the contused area and destroyed the conductivity without penetrating the canal.

These are extremely important cases and in them very little or perhaps nothing is to be accomplished by operation. Sometimes the most profound changes occur in the cord and white matter of the cord without any direct traumatism, and these leave behind them important after-results.

I want to say a few words about the signs of a total transverse lesion of the cord because I am afraid the subject is decidedly misunderstood by the bulk of the profession. In a total transverse lesion paralysis and anesthesia are complete, absolute and permanent. If below the lesion there are at any time contractures or spasms of muscles the lesion is never complete. That is indisputable. If we have a partial lesion we get spasm of muscles, because the vestibulospinal tracts which carry the impulses increasing muscular tone have escaped injury. It is a common mistake to attribute the increased tone and spasm of muscles below the lesion to irritations of the cord produced by displaced bone or blood-clot. If we can hammer into the heads of surgeons the significance of the symptoms of a transverse lesion they will stop operating on these cases. If all men knew exactly what happens when the tracts of the spinal cord are severed they would not operate, because when the tracts of the cord are once destroyed they are never regenerated.

Dr. W. R. Jackson, Mobile, Alabama.—Dr. Ransohoff's paper certainly opens up a very important question. Some of our most aggressive surgeons operate on all cases of broken back. Operations done at the proper time in some cases may do a lot of good. We are taught that immediate paralysis of motion

and sensation below the point of fracture synchronous with the injury means dissolution of the cord. A gunshot wound or violence or fracture dislocation or a stab wound with a knife, with immediate sudden simultaneous disappearance of motion and sensation, means severance of the cord. Violence by force very often results in severing of the cord. The top portion of the vertebral column is knocked forward, the lower portion backward, and if the vertebræ are replaced immediately by muscular action you find nothing, so far as the spinal column is concerned, indicating fracture or dislocation. The *x*-ray picture sometimes shows fracture, as the doctor said. In this connection I would like to report one or two cases.

Three months ago a man fell from a scaffold a distance of 12 feet, struck his shoulder and broke his back. He could not turn over. There was complete abolition of motion and sensation below the fracture. He was carried home and the question of operation was discussed. Some of our enterprising doctors said that operation should be done. I, of course, being conservative, did not operate, and the man died in three days. Possibly if I had operated he might have been living today; I don't think so.

Another case was a boy, aged fourteen years, who in diving hit his head against a sand bar. He was a messenger boy. He went along for three or four weeks with a stiff neck; he had an osteopath and a few other "paths" working on his neck. He finally came to me for investigation. I had an *x*-ray picture taken which showed a dislocation and fracture of the second vertebra, the laminæ of both sides broken in two, and the odontoid process torn loose, almost like a hangman's knot would cause, except it was not sufficiently displaced to press upon the medulla oblongata and kill him. My advice was to let him alone. He was walking around; he had no paralysis but simply a stiff neck. In about three months after the fracture and dislocation of the neck he developed immediately paralysis of the right arm; the next morning the right leg was involved. Consultation was demanded and immediate operation urged. At that time, however, the respiration was involved and there was slight paralysis of motion and sensation on the right side. Laminectomy was done and the fractured piece of the second vertebra, including the spinous process, was removed. The cord was intact. The boy died.

Dr. George A. Hendon, Louisville, Kentucky.—I should like to record a personal experience on account of the connection it had with trophic influences or the trophic phenomena which followed a case of fracture of the third and fourth cervical vertebræ, with complete loss of sensation and loss of motion, from about the level of the nipple down. At the same time the patient sustained a fracture of the right arm below the elbow. She lived about five weeks after the accident, and one of the most prominent features in connection with her case subsequent to the accident was a very strong liability all over the body to pressure necrosis. Wherever a surface came in contact with the bed there was immediately developed necroses and the fracture, which occurred in the arm, made as prompt a union as any fracture I have ever seen. Yet the skin could not bear the slightest pressure.

Dr. Ransohoff (closing).—In regard to the changes of which Dr. Hendon spoke occurring over the body, I consider these among the most important that we have from a prognostic view-point. I cannot understand why it is, for example, that in a man who has a fracture high up, with a complete transverse lesion, in twenty-four hours—and I believe this is the experience of everyone—there will be manifested evidences of trophic changes in the field. I have noticed time and again that within twenty-four hours there would be evidences of gangrene, sloughs about the heels or buttocks or about the scapulæ. I do not know of any other condition, except possibly a gas-bacillus infection, in which this sloughing gangrene occurs with such tremendous rapidity, and for that reason, when we see that, I am quite sure we can count on interference with the trophic centers in the anterior horn, and we can say positively it is a transverse lesion.

In regard to refusing to operate on most of these cases I do not think it should be attempted in cases of injuries to the cervical region in the acute stage, yet there is one case that has been reported again and again in the literature, about fifteen years ago, and I had the privilege of seeing it in the clinic of Schede. The case was one of fracture of the spine with every evidence of a complete transverse lesion, one you would not expect to recover. I saw that man walking about three or four months after operation with a plaster-of-Paris jacket unaided except with a cane. There are some remarkable things in regard

to the possibility of regeneration of the cord. You are familiar with the case reported from Philadelphia in which the cord was resected with complete restitution of function.

With reference to operating if the x-ray shows deformity and the patient survives a certain length of time without improving, why not go in there? I have a case in mind, of comparatively recent date, in which there was paralysis for four weeks of the lower extremity without any indication of transverse lesion of the cord, and after operation motion began in the lower extremities again. That is quite a different proposition from going into an acute case where we have a complete transverse lesion of the cord. I doubt whether a complete transverse lesion of the cord has been benefited by operation, yet patients clamor for operation. It is no trivial matter to operate upon a transverse lesion of the cervical cord.

I am very glad that my paper has elicited so much discussion on this subject. The central thought in my paper was that an x-ray should be taken of each patient with an injury of the spine. If we have evidence of a transverse lesion of the cord, and just before operation is undertaken the x-ray picture shows there is nothing in the bone that keeps up the tissues in the cord, that is not a case on which to operate, but only exploration to determine whether the cord is crushed or not.

A SPECIFIC FOR EVERY PATHOGENIC MICROÖRGANISM

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The idea of a specific in medicine is not new, as both homeopathic and eclectic medicine were built up, at least in part, on this theory. The application of the theory in those instances was, however, premature, as the etiology of disease was not sufficiently understood at that time. These systems attempted to find a specific for each symptom, an attempt which from the very nature of things was doomed to result in failure in a very large percentage of cases. Thus to treat all chills by the same remedy irrespective of their causes is not feasible, because a remedy which is particularly beneficial in a malarial chill, for instance, may be and is, as a matter of fact, very harmful in septic chills.

Today, with our more accurate knowledge of infections, the theory is not only sound but its application in these conditions is feasible. We must, however, not expect the impossible from a specific. We must not expect every patient to be cured. Some cases will succumb in any event. Diphtheria antitoxin comes about as near being a specific as we could wish for, and yet some cases will die from diphtheria no matter how early and carefully the antitoxin is administered. With quinin and other so-called specifics the same is true. Some cases of malaria will go to their doom in spite of every precaution. An occasional case of lues,

particularly Asiatic lues transplanted on a Caucasian, will result fatally in spite of the most expert use of potassium iodide, mercury and arsenic.

Every wild beast has its deadly enemy, every plant its parasite and every parasite its parasiticide; and almost every known chemical poison has its chemical antagonist and probably also its physiological antidote.

Those of us who have an intimate acquaintance with the rattlesnake have a wholesome regard for it, some persons going so far as to believe that it is practically invulnerable; and yet, in addition to man, "the rattler has three natural enemies that are much dreaded by it and which assist in keeping it in bounds—the Mexican eagle (caracara), the hog (wild and domestic) and the speckled kingsnake (Ophibolus getulus)." An old sow will attack, kill and devour a six-foot rattler and make less fuss about it than does a robin when it tackles a large angle-worm. The king snake of Texas will attack and kill a rattler nearly twice its length and more than three times its weight, as vouched for by J. D. Mitchell,[1] of Victoria, Texas, in the following description:

"The king snake (Ophibolus getulus) seems to be able to rob the rattler of its power of resistance, as will be noted in the following account of a battle between them. It was in the spring of 1858, in Calhoun County, I heard the rattler's challenge, or warning rattle, and going to the place I found in a clear space, surrounded by brush and cacti, two very large rattlers, nearly six feet long, each, coiled, with heads up and rattlers sounding. They were looking in the opposite direction from me and seemed more alarmed than angry. Presently a small king snake, not more than half the length of the rattlers, glided out of the brush into the clear space. He came with head and part of his body raised

[1] Transactions of Texas Academy of Science, 1902, v, Part 1, No. 2.

and curved and waving from side to side. He approached the nearest rattler, alternately swelling and flattening his neck, crawled half-way around the rattler and then back again as if looking for an opening. The rattler remained on the defensive, and both he and his mate seemed paralyzed with fear. Finally the king snake made a dart. For a while I could see nothing but a tangle of snakes and a cloud of dust. As the combatants quieted down I saw that the king snake had the rattler by the jaw with his mouth and had his body twisted several times around the rattler's neck. The rattler was striking the ground violently with his tail and the posterior part of his body and the king snake was momentarily tightening his grip by convulsive muscular action. After a time the rattler ceased all motion and the king snake gradually loosened his folds, keeping his mouth-hold on the jaw. Feeling no motion in the rattler, he let go and pushed the rattler's head with his nose, then he crawled over the rattler's body several times, going from head to tail and back again, nosing the head again. Convinced that his enemy was dead, he immediately assumed the offensive attitude and started for the second rattler, which had continued in the same position during the battle with the first. A second battle now followed, the counterpart of the first. When the king snake was satisfied that his second victim was dead, he glided into the bushes and left the dead snakes to me. The king snake was scarcely half the length of either rattler and showed neither fear nor hesitation at any time during the battle. The rattlesnakes showed great alarm from the start, and the second one seemed too much paralyzed by fear to crawl away during the first battle. Neither rattlesnake made a single stroke that I saw. Both rattlers had their heads dislocated from their bodies and several inches of the vertebræ crushed to a pulp."

Every practical nurseryman knows that in order to successfully combat the parasites that attack his trees and

shrubs, he must use the particular spray that has been proved effective for each parasite. For one he will use Bordeaux mixture, for another the Bordeaux and arsenate of lead mixture, for still another the kerosene oil emulsion and for still another hellebore dissolved in water. He would not think of using the same solution for all parasites, for if he did he would not long succeed as a nurseryman. The above illustrations are cited to bring out and emphasize the general laws of nature to which human ailments, particularly infections, are no exception.

The medical man who treats many cases of poisoning would be severely criticized, and justly so, if he should treat all cases exactly alike. While he may use the stomach pump in a considerable percentage of cases, he must use a suitable and different chemical antagonist and a suitable and usually different physiological antidote for each kind of poison in order to be successful in the largest possible percentage of cases.

While it is true that, up to the present time, it has been impossible, so far as I know, to definitely isolate and positively identify the chemical poisons generated by the different septic and saprophytic organisms which cause infections in man, yet no one will doubt for a moment but that they do differ greatly in their chemical nature and also in their toxic action. Anyone who has carefully observed a number of cases of streptococcic puerperal sepsis, with the great nervous irritability engendered by its toxins, and who has also treated a considerable number of severe cases of nasal diphtheria, with the concomitant somnolence and stupor, needs no argument to be convinced that the toxins generated by these two infections must be different. Is it not strange, then, that there are still some physicians who try to treat all infections alike, irrespective of the species of bacteria causing the infection? Thus, Dakin,[2] in a recent article,

[2] Jour. Am. Med. Assn., July 7, 1917, vol. lxix, No. 1.

makes the following statement: "The perfect germicide should meet two simple requirements; it must kill all pathogenic parasitic life, while causing no harm to any cell of the living body." When we consider the different cultural requirements the different pathogenic bacteria have, some preferring alkaline media, others acid media, some growing only when air is excluded, while others must have oxygen, is it not strange that anyone should think it possible to find a universal germicide? To illustrate the unreasonableness of this: Is there anyone today who would expect to cure syphilis, tuberculosis and malaria with the same remedy? To me it is quite as unreasonable to claim that gas bacillus, pyocyaneus bacillus, streptococcus, staphylococcus, influenza bacillus, anthrax, glanders and all the other infections can be cured by one lotion. It is quite as unlikely that a universal germicide will ever be found as that a universal food will ever be discovered for all plants and animals.

Is it not about time that surgeons learn that they too must combat the different infections which attack the human body by using different substances and even by the employment of different methods? In some respects I can see no fundamental difference between the nostrum vender who advertises his cure-all for all inflammations and the physician who himself prescribes and advises others to use the same remedy for all septic and saprophytic infections. The medical press has very justly criticized the former and has reduced his baneful influence to the minimum, but, so far as I know, not a single medical editorial has been written condemning the latter. A certain amount of routine in surgery is unavoidable and may even be of real value, but too much routine is deadly; it kills the enthusiasm of the surgeon, making him a mere human machine and unnecessarily kills some of his patients.

In this connection it must not be forgotten that one drug or remedy may be specific for a number of species of bacteria and also that each species of bacteria may have a number of

specifics; thus boric acid may be looked upon as a specific in streptococcic infections, diplococcus of Weichselbaum infections and pemphigus vegetans, while mercury, potassium iodide and arsenic are considered specifics for the Spirocheta pallida.

As in chemical poisons taken by mouth we usually employ the stomach-tube advantageously, so in septic and saprophytic infections there are certain therapeutic remedies and hygienic measures that are quite universally applicable. Thus a person suffering from either a saprophytic or septic infection should be placed in bed with the affected extremity elevated and a suitable retention apparatus applied so that the muscles of the extremity may be at equilibrium and at perfect rest; proper drainage should be secured by the application of a suitable dressing, or, if macroscopic pus has developed, by carefully planned and skilfully executed incision, being particularly careful not to cut through the leukocyte barrier. If incision becomes necessary one should also be very careful to avoid mixed infection, because while the body has acquired a certain degree of immunity against the active infective agent it has developed no immunity for other infective agents; proper elimination by the skin through suitable baths; proper elimination by the lungs; proper elimination by the bowels, preferably by saline cathartics and suitable food, and proper elimination by the kidneys by diuretics, the best of which is water, should all be carefully looked after.

The balance of this paper will deal mainly with the methods of treatment and definite remedies that have been found by other surgeons as well as by myself as most effective in the various types of infection. Of the many remedies recommended in the literature by other surgeons I have included only those which I have either tried out myself and found effective or which seem reasonable from the stand-point of my own clinical experience. Roughly, I will consider the more common infections under two headings: (1) Those

in which, so far as I have been able to ascertain, no real specifics have been discovered, though even among these I will cite some forms of treatment that have been highly recommended by reliable observers, and (2) those in which it would seem that our clinical experience justifies us in the conclusion that certain substances may be looked upon as true specifics in certain definite infections.

Anthrax and Glanders. With anthrax and glanders I have had no personal experience and, so far as I know, no specific has been discovered for either.

Influenza Bacillus. Though I have treated two cases of pure influenza bacillus peritonitis, the remedies which I tried were futile and both patients succumbed to their infection. I have also treated quite a number of cases of apparently pure influenza sore-throat, and here too the local applications that I tried were of no particular value. In my perusal of recent literature I have been unable to find any remedies which seem able to inhibit this infection.

Gas Bacillus. I have only seen a few cases of gas bacillus infection, and these were seen either in the postmortem room or through the courtesy of some other surgeon. None of them have been under my personal care, so that I cannot speak from personal experience. Davis[3] suggests long, deep parallel incisions which are joined together subcutaneously; the field is then rendered aërobic by the injection of oxygen. Taylor[4] found malic and tartaric acids and quinin hydrochloride very potent in inhibiting the growth of gas bacillus in culture media. Later he[5] tried quinin hydrochloride, in one-tenth of 1 per cent. solution, in normal saline for the purpose of irrigating well-incised gas infections, with good results.

Blastomycosis. In blastomycosis my personal experience is rather limited, but, so far as it goes, potassium

[3] Jour. Am. Med. Assn., June 9, 1917, vol. lxviii, No. 23.
[4] London Lancet, February, 1917, vol. cxcii, No. 4878.
[5] Canada Lancet, April, 1917, vol. l, No. 8.

iodide internally and very careful excision, being careful not to reinfect the wound, seem to be the most effective remedies.

Acute Articular Rheumatism. While the specific microbic cause of acute articular rheumatism has not been definitely determined, I believe that 60 grains of sodium salicylate dissolved in 8 ounces of normal saline, and given by rectum once or twice a day, as first suggested to me by Dr. Emil A. Ochsner, of Rockford, Ill., comes very nearly being a specific. It is certainly true that sodium salicylate given in this way is much more effective, and that in much smaller doses, than when given by mouth. In addition, the joints should be carefully immobilized, with the muscles at equilibrium, and acids and meats should be excluded from the diet. I have included acute articular rheumatism in this list because I believe proper immobilization is one of the most important elements in its successful treatment, a therapeutic remedy rarely properly employed by internists.

Colon Bacillus. In some conditions colon bacillus is a very intractable infection, but in my experience most cases have responded to the administration of autogenous vaccines. I have found from 5,000,000 to 25,000,000 dead bacilli given once or twice a week to be more effective than larger doses. If the colon bacillus is lodged in a superficial wound, such a wound as occurs after a pus appendix operation, I have found daily tub baths at 93° F. for one-half hour very effective in hastening the healing.

Gonorrhea. Probably in no other infection has there been greater effort made to find a specific than in gonorrhea, but so far as I know none of the remedies which have been so highly recommended from time to time have stood the test of clinical experience. Silver salts are still in favor with many. Personally I have had the best results with vaccines, but I believe I have had better results with the smaller doses beginning with 5,000,000 of the dead gonococcus and gradually increasing to 25,000,000, giving these injections with intervals of from five to seven days.

Chancroid. In chancroidal infection, Dr. William A. Pusey considers iodoform a specific.

Specific Inguinal Adenitis. Many methods of treatment have been recommended for this condition, and, while these methods are fairly satisfactory, I have seen a number of cases of specific inguinal adenitis treated by these various methods that had sinuses remaining weeks, months and even years after the original incision or other forms of treatment had been instituted. When cases have come to me before they have been incised or before they have ruptured I have had surprisingly good results by making a large elliptical excision of the whole infected area, being careful not to cut into the infected foci, then pack the wound for a few minutes with a strip of gauze soaked in Churchill's tincture of iodin, then apply a dry dressing for four days, then a daily tub bath at 93° F. for one-half hour, with dry dressings during the interval. When the granulations are firm, usually at the end of about a week, I put on a dry skin graft,[6] and in that way have always secured complete healing in a relatively short time. This method has been uniformly satisfactory in my experience, and materially shortens the period of healing in most cases.

Syphilis. I wish to insert syphilis here principally as an illustration of the value of specific treatment without making an attempt to go into details, which can be found in the monographs on this subject, many of which recommend 33.3 per cent. calomel ointment as a prophylactic; neosalvarsan every three to ten days until six doses are given, just as soon as a diagnosis can be made by finding the Spirocheta pallida in the initial lesion; mercury, potassium iodide and neosalvarsan in the secondary, tertian and quartan stages.

Malignant Edema. For malignant edema, Lord Lister[7] advised "scraping away very thoroughly under chloroform

[6] Ochsner: Chicago Med. Recorder, December, 1900.
[7] Third Huxley Lecture, Harrison & Sons, 1907.

the brown pultaceous slough and freely applying acid per nitrate of mercury to the exposed surface, then applying a poultice until the eschar has separated." Fenger recommended infiltrating the healthy tissue all around the infected area with glacial acetic acid, in this way securing mummification of the diseased area, this to be followed by excision or high amputation. Lawrence Ryan recommends high amputation, with eversion of flaps, treating the wound with hydrogen peroxide and injecting the tissue with pure oxygen.

For balanitis gangrenosa, probably a form of malignant edema, Pusey[8] recommends slitting the prepuce wide open, exposing the parts to the air and keeping it bathed with 2 per cent. peroxide of hydrogen.

Noma. In two cases of noma which I treated in conjunction with Dr. Lawrence Ryan, at the Cook County Hospital, we found bromin fumes very effective.

Impetigo Contagiosa. Personally I have found a 5 per cent. yellow oxide of mercury ointment or a 3 per cent. ammoniated chloride of mercury ointment very effective in this condition. Morrow[9] recommends to squeeze the pus out of the individual pustule, remove the scales and dead skin with epilating forceps, swab the eroded bases with 20 per cent. silver nitrate solution and then dust the surface with a dusting powder consisting of ammoniated chloride of mercury, 6 to 10 per cent., boric acid, 15 to 20 per cent., and talcum sufficient to make 100 per cent. Dr. Lawrence H. Prince, school physician of Madison, Wis., in a personal communication, told me that he had found the daily application of spirits of camphor to each pustule very effective. If the children are willing to submit to the application daily, he found it unnecessary to exclude them from school. I have included impetigo contagiosa here because I have seen a number of cases of this condition treated by surgeons with

[8] Jour. Am. Med. Assn., September 29, 1917, vol. lxix, No. 13.
[9] Ibid., July, 1917, No. 3.

wet dressings, evidently on the supposition that it was an ordinary infection, with the result that the process kept on spreading for many days, in one instance until the patient finally went to another surgeon, who applied yellow oxide of mercury and secured healing in a few days.

Rodent Ulcer. So far as I have been able to determine, pathologists generally look upon rodent ulcer as a type of carcinoma. With this view I cannot agree: (1) Because there is too much round-cell infiltration; (2) because to me the cells looked upon by pathologists as true carcinoma cells do not look exactly like cancer cells; (3) because we do not get the same kind of regional lymph involvement; (4) because metastasis in rodent ulcer is practically unknown; (5) because it does not show the same tendency to recur when properly excised. Wide excision, being careful not to infect the operative wound, with subsequent plastic or skin graft, has resulted in complete and permanent cure in every case of rodent ulcer that has come under my care.

Actinomycosis. In actinomycosis, removing the foci of the infection, such as diseased tonsils, teeth, beards of barley or chaff of grain, then laying open the sinuses to the very tips and giving the patient 60 grains of potassium iodide for three days, followed by an interval of four days and then repeating, as first suggested by veterinarians in the treatment of "lumpy jaw" in cattle, has resulted in cure of every case which I have had under treatment. It is quite essential that all the above things be done carefully and conscientiously. I have seen a number of cases in which one or the other of the above directions was neglected. The disease persisted for months, and was only relieved when they were all systematically employed. Giving the potassium iodide in large doses and then an interval of suspension of the drug seems quite an important item in securing prompt and permanent relief.

Surgical Tuberculosis. There are a number of very important points in the treatment of surgical tuberculosis.

Probably the most important of these is the prevention of secondary infection. The others are principally fresh air, proper food, rest and vaccine therapy.[10] I have found minute doses of vaccine, from 0.000075 mg. to 0.0002 mg. of the dry, new, tuberculin residue given hypodermically once or twice a week much more effective than larger doses. For joint tuberculosis,[11] complete and absolute rest for a considerable period of time, with the other items well looked after, will result in a healing of the process in almost every case, with motion of the joint reëstablished in a very considerable percentage.

For tuberculous ulcers without sinus formation I have recently been applying a saturated solution of picric acid. I apply it with a cotton swab every second or third day, being careful to get as little of the solution on the epidermis as possible. Then I cover this with a dry dressing held down snugly to avoid mixed infection. This treatment seems to favor the healing greatly.

Rabies. For dog, cat, wolf or skunk bites the bitten area should be rendered surgically clean, then under anesthesia the wound should be laid wide open and packed with a piece of gauze saturated in Churchill's tincture of iodin, leaving it in for ten to fifteen minutes, even at the risk of slight vesication. After this a 50 per cent. alcohol dressing should be applied. The biting animal should be kept in restraint and carefully observed, and if rabies develops, Pasteur treatment should be immediately instituted. If the biting animal has been lost track of and there is the slightest suspicion that it is rabid, Pasteur treatment should be immediately resorted to, because when rabies has once developed, treatment is, so far as we now know, futile.

Tetanus. If there is any likelihood that a puncture wound, gunshot wound, compound fracture or any other

[10] Ochsner, Edward H.: Illinois State Med. Jour., February, 1909.
[11] Ochsner, Edward H.: Michigan State Med. Jour., August, 1908.

accidental wound is contaminated with tetanus bacilli the patient should be anesthetized or the surface rendered anesthetic by the application of 3 per cent. carbolic acid in water for three to five minutes. The area should be rendered surgically clean; an Esmarch applied wherever possible; the wound laid wide open; all foreign bodies, such as clothing, paper wads, loose fragments of bone, bullets, etc., carefully removed; then the wound should be carefully swabbed with 95 per cent. carbolic acid, which should be left in place for a few moments, then washed out with 95 per cent. alcohol, the Esmarch removed, all bleeding-points carefully ligated, the wound dressed wide open with gauze saturated in 50 per cent. alcohol, then an immunizing dose of antitetanic serum should be immediately given. By following the above routine in every case I have not had a single case of tetanus develop in sixteen years, though one of my cases developed trismus on the eighth day and one trismus and opisthotonos on the twelfth day.[12] In the former case symptoms only lasted about eight hours and the latter only forty-eight hours. In neglected cases where tetanus has developed, Burrows[13] recommends injecting 3000 units of antitetanic serum into the muscles of the infected region, 3000 units into the spinal canal and 3000 units intravenously.

Erysipelas. Erysipelas I consider a self-limited disease, which will get well in the great majority of cases provided the general hygiene and elimination are looked after properly, and the treatment is not too heroic and drastic. In 47 consecutive cases treated at the Cook County Hospital during March and April, 1895, by the use of various external applications I lost only 1 case. This case was a compound fracture of the skull, and was infected by the operating surgeon who, himself, was coming down with erysipelas

[12] Ochsner, Edward H.: Medicine, June, 1906.
[13] London Lancet, January, 1917.

without his knowledge. This patient died from meningitis. In private practice I have never lost a case, though one case was complicated with pneumonia, one with delirium tremens and a number occurred in very old patients. Of all the various applications tried I have found that a wet dressing, consisting of saturated solution of boric acid in water, with from 15 to 33.3 per cent. of 95 per cent. alcohol, the most comfortable to the patient, and I am inclined to think a little the most effective in aiding the production of immunity and hastening convalescence.

Streptococcus, Staphylococcus Albus, Staphylococcus Citreus[14] and Diplococcus of Weichselbaum.[15] In all strains of streptococcus, Staphylococcus albus, Staphylococcus citreus and diplococcus of Weichselbaum I have found boric acid in water with alcohol in the proportions above mentioned more satisfactory than any of the other remedies tried. In fact, I look upon this wet dressing, if properly applied, as a true specific for the above infections. If the infection is superficial, first painting the reddened area with 95 per cent. carbolic acid until it turns white and then washing this off with 95 per cent. alcohol will almost instantly relieve the pain, destroy myriads of bacteria and considerably hasten recovery. Powell[16] describes a very interesting case of extensive destruction of vulva by practically pure pneumococci infection, which was successfully treated by painting the area with 95 per cent. carbolic acid, then alcohol, dusting the surface with equal parts of boric acid and iodoform. From my experience with the diplococcus of Weichselbaum I am inclined to believe that the satisfactory outcome was due largely to the use of boric acid.

Pemphigus Vegetans. For pemphigus vegetans I consider a saturated solution of boric acid in water as a wet

[14] Ochsner, Edward H.: Transactions of the Southern Surgical and Gynecological Association, 1916.

[15] Ochsner, Edward H.: Illinois Med. Jour., February, 1917.

[16] Jour. Am. Med. Assn., April 10, 1915, vol. lxiv.

dressing a specific, and while my conclusion is based on only two cases, the cases were so clear and convincing that I think I am justified in my conclusion. During March and April, 1895, I was in charge of the contagious ward at the Cook County Hospital. My attending man was the late Dr. D. D. Bishop, a former assistant of Drs. Hyde and Montgomery. During this period a very sick man came under our care. Dr. Bishop, after a very careful examination, made the diagnosis of pemphigus vegetans, and expressed it as his opinion that the man could not recover. As it was the period of my early investigations in the use of boric acid I suggested that he permit me to try a saturated solution of boric acid on the case. To this he readily consented. I gave the patient a tub bath of boric acid daily, using a couple of gallons of the solution in a bath-tub, and also had him sponge the involved surfaces with the solution. Then I swathed every portion of the body that I could in dressings moistened with saturated aqueous solution of boric acid, and to our great surprise the patient made a rapid and complete recovery. The patient was a charity case, was lost track of, and whether he succumbed to a subsequent attack I have no way of knowing. Dr. Bishop, in his characteristic modest way, later doubted his own diagnosis, because he said all authorities agreed that patients suffering from the type and severity of the disease presented by this patient invariably succumbed to the disease. In the summer of 1898 a patient with pemphigus vegetans presented himself at the Augustana Hospital. I felt very positive of my diagnosis, but to have it corroborated I took the patient to the late Professor Hyde, who agreed with my diagnosis and told me that the patient would shortly die from the disease. I put him on the same treatment as the one above. The patient made a rapid and complete recovery, and Prof. Hyde was no little surprised when I took the patient to him several weeks later. He asked the privilege to show him at the Alumni Clinic at Rush Medical College,

stated before that clinic that he had never seen a man suffering from that affection to the degree in which this man suffered, recover even temporarily, but expressed it as his further opinion that the man would have subsequent attacks from which he would undoubtedly succumb. I told the man his great danger, but urged him to submit to the same treatment if ever he had a recurrence. About ten years later the patient came to my office, told me that he had had half a dozen recurrences, each one less severe than the previous one, and each one treated in the same way, and that for the past four or five years he had been entirely free, and that since then he had been in perfect health. Careful physical examination corroborated the latter statement.

Staphylococcus Aureus. In Staphylococcus aureus I find that boric acid is not nearly as effective as in the previous mentioned infections. I find that Staphylococcus aureus is usually the infective agent in carbuncles and furuncles, and I further find that aqueous wet dressings, as well as tincture of iodin, are very apt to favor the development of new crops of carbuncles and furuncles. For furunculosis I have found painting each furuncle with 95 per cent. carbolic acid, then applying a wet dressing of 50 per cent. alcohol or a 5 per cent. yellow oxide of mercury salve most effective. Before this is done, however, it is well to take a culture and make an autogenous vaccine, so that if the case should prove to be stubborn one can inject from 5,000,000 to 25,000,000 dead staphylococci once or twice a week. Here, again, I have found small doses of vaccine much more effective than large ones.

The above method of treating furuncles is particularly to be recommended and urged in furuncles of the nose. Here the central hair should first be epilated, then the furuncle treated with 95 per cent. carbolic acid on a probe armed with a small cotton pledget, this neutralized with 95 per cent. alcohol, and then 5 per cent. yellow oxide of mercury

applied. Because of the direct communication of the mucous membranes of the nose and the superior longitudinal sinus, excising furuncles of the nose is extremely dangerous. I have known a number of patients to succumb, who were treated by excision, who would probably have gotten well with little difficulty if the treatment above recommended had been employed instead.

In carbuncles I have found injecting 95 per cent. carbolic acid into the base of the carbuncle by far the most effective method of all tried. If the carbuncle is less than 2 inches in diameter, introduce the needle at the margin of the inflamed area, inserting it until the point is located directly under the base of the carbuncle, then inject 95 per cent. carbolic acid until it oozes through the apex of the carbuncle. I then apply a wet dressing of 50 per cent. alcohol and later 5 per cent. yellow oxide of mercury ointment. The injection of carbolic acid usually relieves the pain very rapidly, results in early exfoliation of the core and leaves a granulating surface which usually heals very rapidly. Only recently a man came to me with a very painful carbuncle of the neck, fully 1½ inches in diameter, who experienced almost complete relief from pain within a few minutes after carbolic acid was injected; the wound was practically healed and needed no further dressings after the eighth day. If the carbuncle is over 2 inches in diameter I make a number of punctures, making sure that every part of the necrotic tissue is thoroughly treated with 95 per cent. carbolic acid. Wherever possible I take a culture and make an autogenous vaccine, which may come in handy if the case should not be so rapid in recovery. I believe I have been able to make the observation that cases treated by the above method are much less likely to have subsequent crops of carbuncles. In a few cases, when the carbuncle has been very large, I have excised it completely with a knife, sometimes with excellent results, and a number of times the cases have resulted fatally, though in the latter instances they have

always been complicated with diabetes, and the unfavorable outcome may easily have been attributed to that condition. Dr. Vilray P. Blair, of St. Louis, Mo., in a personal communication, told me that he has had splendid results in excising carbuncles with the electric cautery. I can see how this can be a very satisfactory method of treatment for a large carbuncle, but for smaller carbuncles the injection of carbolic acid does away with a general anesthetic and causes less pain, and I believe results in more rapid recovery as a general rule.

Chronic Sepsis. In neglected cases of chronic sepsis, where the wound has or has not healed, but where the patient does not seem to be able to gain strength, I have found hot-air baths, with profuse sweating and tub baths at 93° F. on alternating days, very beneficial. Hoelcher[17] has demonstrated that profuse sweating will remove considerable detritus and aid in the elimination of much toxic material. I have applied this observation in a good many of these cases of chronic sepsis, utilizing the hot-air baths to eliminate the toxic material from the system and the tepid baths to stimulate the skin and soothe the nervous system, and have had the satisfaction of seeing some of these apparently hopeless cases get a new start which ultimately lead to their recovery.

Pyocyaneus. For over twenty years I have been looking for some remedy to overcome pyocyaneus infection and have made use of nearly every antiseptic I could think of. When formaldehyde first came into use I tried it in various strengths, even going to the trouble of rigging up an apparatus to generate formaldehyde fumes. Later, remembering the high repute vinegar had at one time enjoyed, I used vinegar dressings. None of the remedies which I employed seemed to be of any special value, and sodium bicarbonate

[17] New York Med. Jour. and Philadelphia Med. Jour., consolidated for February, 1904.

dressings actually increased the amount of pyocyaneus pus. Evidently I did not use the vinegar in the right strength, because recently Taylor[18] reports excellent results with 1 per cent. acetic acid in normal saline solution. He found that Bacillus pyocyaneus is rarely if ever found in strongly acid purulent secretions, so in trying various antiseptic dressings he discovered that acetic acid was practically a specific in the treatment of pyocyaneus infections. That the beneficial action of the acetic acid solution is not entirely dependent upon its acidity is proved by the fact that nitric and hydrochloric acids in weak solution had little inhibitory effect, and we all know that boric acid solutions do not inhibit its growth. Sodium bicarbonate seemed to stimulate the growth of the pyocyaneus bacillus, as did also Dakin's solution. Since reading Taylor's article I have had only 2 cases of pyocyaneus infection. One was relieved by applying the dressing twice and one by applying it once.

Saprophytic Infections. In indolent ulcer, particularly non-syphilitic ulcer of the leg, I have found Unna's paint, applied in the form of an Unna's paint boot, as first introduced in this country by Dr. W. S. Royce,[19] very satisfactory. By this treatment I have been repeatedly able to completely heal large non-syphilitic ulcers of the leg, with the patient at work nearly every day. For some reason syphilitic ulcers do not tolerate this dressing well, even if the patient is placed on active antisyphilitic treatment during its application. I have had a number of cases where the pain from the dressing was intolerable and the boot had to be removed. In these cases I have found a remedy suggested by Dr. Stuart McGuire[20] very beneficial. It consists of a wet dressing of 2 grains of chloral hydrate to each ounce of water, the dressing being kept moist constantly. This dressing, however, must be used without a rubber protective.

[18] Jour. Am. Med. Assn., November 25, 1916, vol. lxvii.

[19] Illinois Med. Jour., 1904.

[20] The Profit and Loss Account of Modern Medicine, p. 266.

Since using this dressing I have repeatedly been surprised at the rapidity with which these old ulcers will epidermicize. In my experience it has proved much more valuable than scarlet red, and I believe the remedy is not only of value for indolent ulcer but for all forms of saprophytic infections. Dr. H. M. Richter, at a recent meeting of the Chicago Surgical Society, recommended continuous tub baths for saprophytic infections. Many others are now using Dakin's solution and nascent sodium hypochlorite for this class of cases. Personally I have for the past thirteen years repeatedly prescribed daily tub baths at 93° F., leaving the patient in the bath for about half an hour. This treatment has often proved very beneficial.

Mixed Infections. Mixed infections sometimes tax the surgeon's ingenuity to the limit, principally because it is often difficult to isolate the infective agents and thus to determine upon the most suitable remedies. If the infective agents are a parasite and a saprophyte, treating the wound with the indicated parasiticide and then with a remedy best suited to overcome the saprophytic infection usually accomplishes the desired result. In mixed infections, where one of the infective agents is the tubercular bacillus, Beck's bismuth paste has proved of real value. In other mixed infections, where I have tried it, I have not found it of any particular value—in fact, I have always found some one or more of the remedies heretofore numerated very much more effective.

I have presented the foregoing conclusions at this time because I believe the personal experience of the members of this Association would be a valuable contribution to surgery, and because there is some danger at this moment of having all of this knowledge put temporarily into the discard by a new fad. At the beginning of the present war the lay press informed us, with flaring headlines, that Dr. Carrell was going to accomplish wonders by replacing lost ears and noses and faces by new ones and thereby was going to

revolutionize the art of surgery and minimize the horrors of war. However, something must have gone wrong. At any rate, very suddenly this class of copy disappeared from current literature. Something had to be done to fill space and a new eighth wonder had to be discovered to appease the American public's desire for sensation. A new solution had to be invented. For the sake of euphony this solution was called "eusol," and while I believe this remedy is of real value in some saprophytic infections, and possiby also in certain types of septic infections, particularly some neglected cases of mixed infection, such as the severely mutilated war shrapnel wounds, I am thoroughly convinced that it is absolutely useless in some cases of septic infection, and even positively harmful in others, as in the following case, the history of which was furnished me by Dr. H.:

"On about March 14, E. B., aged twenty-one years, called at my office complaining of a painful callus over the insertion of the right tendo Achillis, caused by a shoe. I painted it with iodin, made free incision and dressed with wet dressing of bichloride of mercury, 1 to 5000. I was called to see him early next morning and his family reported the patient had had a very restless night, with a hard chill about midnight, lasting one-half hour. The leg was swollen up to the knee and very painful. I sent the patient to the hospital, and under an anesthetic made a number of free incisions, dressed with Dakin's solution twice daily for about ten days, temperature remaining 102° to 104°, and the patient becoming sicker each day. You were then called in consultation and advised saturated solution of boric acid, 3 parts with 1 part of 95 per cent. alcohol. Within twenty-four hours we had a changed picture: Temperature was lower and the patient's general condition appeared much better. He continued to improve, and at the end of a week his temperature was normal and the infection had subsided."

When I saw the patient he was desperately sick, the whole leg up to the knee was greatly swollen, tender and very

painful. The subcutaneous cellular tissue was boggy and pus was discharging from numerous wounds.

Taylor[21] also found Dakin's solution nearly inert in pyocyaneus and gas bacillus in culture media, and actually found it to increase the growth of pyocyaneus bacillus on wound surfaces. He also found it lacking in bactericidal power on the Bacillus aërogenes capsulatus and the Staphylococcus aureus, both in culture of broth and a medium containing albumin. O'Connor[22] found eusol useless in cases of infection caused by the gas bacillus and streptococci. In this connection it must be remembered that while cultural investigation as to the value of a certain remedy in the treatment of certain types of infection has its value, it must not be relied upon exclusively, but must always be controlled by clinical experience and observation. Thus while it is a well-known fact that boric acid[23] does not inhibit the growth of pus microörganisms it does reduce their virulence and greatly assists the body to overcome their deleterious effect. Taylor has demonstrated that while cresol in 0.08 per cent. solution will inhibit the growth of Bacillus pyocyaneus in culture media it has little or no effect upon the development of Bacillus pyocyaneus when applied to an ulcerative surface infected by this organism.

In an article by Dakin,[24] Lee, Sweet, Hendrix and Le Conte is the following quotation: "After careful surgical preparation of the wound at the primary dressing and excision of all foci of infection and of devitalized tissues," etc. While this may be good advice in the case of some war wounds which come to the surgeon later than twelve hours after their infliction, it would certainly be bad advice in the case of ordinary septic infections of civil life. Few civil surgeons of large experience would, I am sure, be willing to follow

[21] Jour. Am. Med. Assn., November 25, 1916, vol. lxvii.
[22] Annals of Surgery, April, 1917.
[23] Loc. cit.
[24] Jour. Am. Med. Assn., July 7, 1917, vol. lxix.

this advice as routine practice for the simple reason that the foci of infection may be so numerous and involve such vital anatomical structures that excision of all of the foci of infection and of all devitalized tissues would be incompatible with the life of the patient. Take, for example, cases of severe pelvic infection or abscess of the lung or multiple abscesses of the liver. There is another serious objection to this procedure in civil practice, and that is, if this advice were followed the incision would have to be made beyond the line of demarcation, which would necessarily open the lymph channels to infection, would tend to cause general sepsis and unnecessarily delay healing of the wound. Take as an illustration a circumscribed infection of a finger which usually will recover in a few days by applying the right dressings and securing rest. Excising such an infection would be almost sure to cause the infection to spread along the tendon sheaths, which would cause permanent contracture and would certainly greatly lengthen the period of convalescence, to say nothing of the increased risk from sepsis.

We should be very careful not to become faddists, because a faddist in medicine is always a nuisance, and sometimes even a serious menace to the people whom he serves. While it is possible that eusol or Dakin's solution may be of real value in treating the horribly mangled shrapnel-wound victims, I am thoroughly convinced that if the attempt is made to indiscriminately substitute it in the treatment of septic infections that occur in civil life much serious damage may be done and many an honest, hard-working, American workman will, in the course of the next few years, unnecessarily acquire contractures, incapacitating him for life; many will unnecessarily lose their limbs and some even their lives. These unfortunate results can be avoided if we will treat each case of septic infection with the remedy which has been found most effective during many years of painstaking study, investigation and clinical experience

This is in part the evidence and the argument in support of my contention that we should make persistent effort to find and then to apply a specific remedy for every pathogenic infection. If we do this we may be confident that the results in the future will be much better than they have been in the past, and infinitely better than if we continue to jump from one fad to another and treat all infections by the one or two remedies which happen to be the fashion.

TRAUMATIC BRAIN SURGERY

By John Wesley Long, M.D., Major, M. R. C., U. S. A.
Greensboro, N. C.

Trauma of the brain is by no means a new subject. It is as old as the mark set upon the brow of Cain, the first murderer. Notwithstanding its antiquity, its importance was never so great in the history of the world as it is today. Therefore, it behooves us to learn all that is knowable regarding the subject in order to prepare ourselves for the task set before us.

My message today is especially to that large number of Southern surgeons who are being called from civil to military service. I would have them go to their new field of activity with that self-same confidence characteristic of the American hero, whether professional or in the ranks. They should know that although they go among strange peoples, speaking foreign tongues, their work as applied to brain surgery involves the same principles which they have been accustomed to follow in treating cracked skulls in railroad camps and pistol-shot wounds of the head due to the time-honored custom of toting a gun in one hip pocket and "moonshine" liquor in t'other.

Major M. G. Seelig, the editor of *War Surgery*, says in the preface:

"The use of the phrase 'war surgery' must be taken with a great deal of qualification, lest one fall into the error of thinking of this type of work as separate and distinct from the surgery of civil life. As a matter of fact, the surgical prin-

ciples governing both are in large part exactly the same. The laws of ballistics, trench life, the terrain of the battle field, the problems of transport and numerous other incidentals serve to modify established principles of surgery, but not more than that."

If my own conclusions are worth anything, traumas of the brain in military practice resemble very closely the same class of injuries which I have had occasion to observe in thirty-six years of civil practice except in so far as they are modified by the character of the missile and the environment. I am sure the same conviction is held by many of my audience. Every civilian general surgeon, especially in the South, is more or less familiar with head traumas and gains the wisdom of experience in proportion to the cases which have come under his care.

I recently heard a distinguished surgeon, who has seen much war service, deprecate information touching war surgery that was gained in any other way than by personal observation at the front. He spoke of knowledge gleaned from medical journals, reviews, etc., as a "correspondence course." However, if we all had to depend alone upon personal observation at the front the 14,000 civilian surgeons who are already in the Medical Reserve Corps and the 8000 more who will soon be in service would go to their task poorly prepared and with no adequate conception of the high duties that await them.

In the preparation of this address I have not hesitated to draw from every available source at my command. In this I have been fortunate. About two hundred articles on skull and brain surgery alone have been published since the beginning of the war; several comprehensive reviews on traumatic brain surgery, notably Eisendrath's, have recently appeared; the War Department has issued a monograph of *War Surgery of the Nervous System*, and the Clinical Congress of Surgeons, just held in Chicago, was distinctly a war meeting, with a number of papers by eminent specialists

in brain surgery. I attended that meeting for the special purpose of taking notes to be used on this occasion.

Those of us who are going into the service to do brain surgery should not neglect any opportunity to prepare ourselves to the utmost for the responsibilities of the work. We should review our anatomy and physiology. Gray's *Anatomy* contains some interesting reading at this stage of the game. We should saturate our minds with the recent literature of the subject, which, by the way, includes all kinds of contradictions regarding antiseptics as well as other things. Whenever possible everyone interested should take advantage of the intensive training generously provided by the Surgeon-General.

I have recommended to the Surgeon-General over three hundred physicians for commissions in the Medical Officers' Reserve Corps. Occasionally some young or middle-aged doctor, never an old man, objects to the military training in the cantonments. I usually tell him the story of Grant and his coon dog: When Grant was a boy he had a pup that he wanted to train to hunt coons. He persuaded his old father to drag a coon skin around the yard for the pup to trail. Being an overindulgent father, like most of us are, the old man dropped on his all-fours and started off with the coon skin dangling from his neck. As soon as he was out of sight the boy turned the pup loose and sicked him on the trail. The pup soon caught up with the old man and fastened his teeth in his shoulder. When Grant reached his father he was yelling for him to pull the dog off. Young Grant said: "Hold on, dad; I know it hurts, but it's the makin' of the pup."

As you know, every regiment has its own surgeons. Seven are usually allotted to each regiment. The scarcity of surgeons in Europe has been so great that only one surgeon each could be supplied to many regiments. Let me say here that there is not a more important man in the Medical Corps than the regimental surgeon. He knows the men and they

come to know and rely upon him. He eats the same food, sleeps with them, goes into the trenches and over the top with them, and if he is the right kind of man the men fight all the better for knowing that he is at hand to succor them if wounded. Soldiers are said to have a great dread of bleeding to death, and well they may. The morale of most of us would be better if we knew that a good surgeon was within a few feet of us. All honor to the regimental surgeon!

It is here, too, that the ambulance companies get in their splendid work. I am pleased to recall that I was largely instrumental in organizing a Red Cross Ambulance Company at Greensboro composed of five as fine surgeons and one hundred and nineteen splendid men as ever went to France.

Since we are concerned only with brain injuries we shall eliminate all discussion that does not bear directly upon that.

When we remember that 40,000,000 men are under arms, not to speak of the unnumbered millions of civilians within the war zone subject to injury by both land and sea, from the earth beneath and the heavens above, one ceases to wonder that brain injuries are both numerous and fatal. The first American officer to shed blood upon French soil was wounded in the head October 29, 1917.

Trench warfare protects all portions of the body better than it does the head. It is true that the steel helmet has lessened both the frequency and the severity of brain injuries. This protection is augmented by the blanket padding employed by the English. Notwithstanding every precaution, about 10 per cent. of the total casualties are of the head.

There have been many classifications of brain injuries. The following is simple and comprehensive:

CLASSIFICATION OF BRAIN INJURIES

1. Brain traumas without visible wounds.
2. Scalp wounds with no apparent fracture.
3. Tangential or gutter fractures of the skull.

4. Depressed fractures of skull from direct blows.
5. Penetrating wounds of brain classified as:
   (*a*) Punctured wounds as from a bayonet.
   (*b*) Missile, as bullet, lodged in brain.
   (*c*) Through-and-through wounds.

High explosives play an important part in brain injuries. Leriche reports in detail 99 cases of severe concussion of the brain and spinal cord in which there was no direct wound. He also saw 397 cases in which a shell fragment merely bruised the scalp. In these cases brain lesions were so frequent that he now operates in all cases. Temoin, in 33 operations for slight injuries, found fracture of the skull in 29. Of 23 such cases operated upon as soon as they reached the hospital, all recovered; of 5 similar cases in which operation was deferred until brain symptoms developed, 4 died. The experience of these surgeons is that of many others. The following cases are from my own practice:

Fracture of Skull—Late Symptoms

Patient: R. L. Johnson, Concord, N. C., aged eighteen years.

Trauma: Caused by baseball striking right side of head.

Symptoms: Headache. Patient fell, got up, seemed rational, returned home; next day complained of headache, which continued.

Coma: On the fourth day developed semicoma, which increased to complete coma.

Convulsions: On seventh day had three general convulsions.

Operation: Craniotomy, eighth day, right temporoparietal region, October, 1898.

No wound of scalp nor fracture of external plate. Internal plate fissured; large extradural clot, smaller intradural clot.

Result: Prompt recovery, healing by first intention; no subsequent nervous symptoms.

Destruction of Entire Half of Brain

Patient: Thomas Willis, Walnut Cove, aged twenty-six years.

Trauma: Patient was struck on head by foul ball.

Case History: Patient knocked down; arose presently, walked one-half mile to home, told wife he was going to die. Ate supper, went to bed complaining of headache. During the night developed convulsions, semicoma and by morning complete coma.

Transportation: Next morning was carried forty miles to St. Leo's Hospital by Dr. A. G. Jones.

Examination: Patient comatose; pupils dilated; pulse frequent and feeble; limbs and back rigid from general spasticity; frequent convulsions.

Head: No wound of scalp or skull could be found.

Result: Patient died without operation four hours after admission and thirty hours after injury.

Autopsy: No wound of scalp except slight bruising in right temporal region. Outer plate of skull intact. Linear fracture of inner plate; no depression. Right meningeal artery ruptured. The entire right half of brain like scrambled eggs.

Fracture of Base; Late Operation (*see Fig.* 1)

Patient: Charles Rayle, aged thirty-one years.

Trauma: Patient knocked to cement walk by awning rail.

Symptoms: Consciousness, slight at first, quickly disappeared.

Convulsions: Three general convulsions first twenty-four hours.

Headache: Slight at first, gradually increased to unbearable degree.

X-rays: Fracture involving left lambdid suture.

Operation: Wesley Long Hospital, August 25, 1917. Craniotomy seven days after trauma; removed bone 1½ by

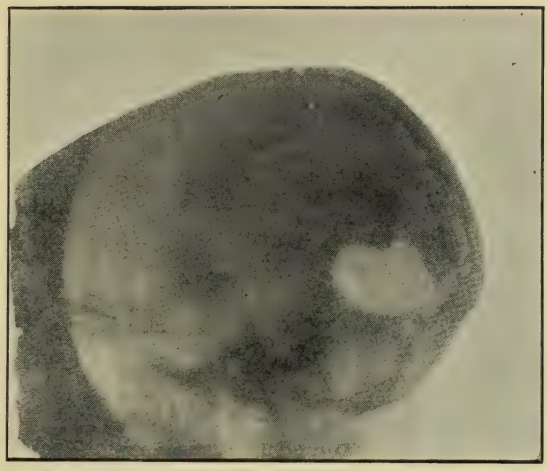


Fig. 1.—Fracture at base of skull; rupture of left lateral sinus; trephined; recovery.

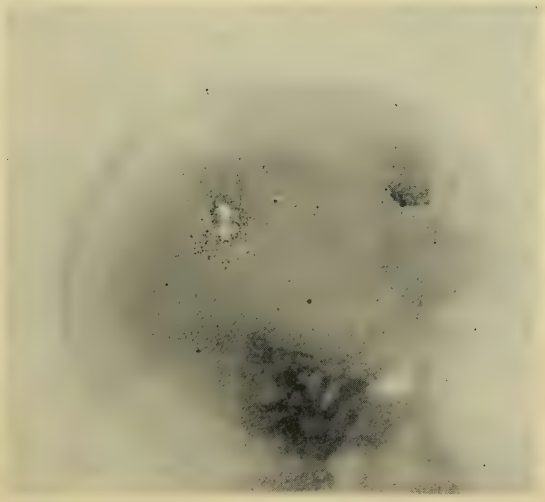


Fig. 2.—Fracture of skull; perforation of brain two inches with pick; abscess; trephined; recovery.

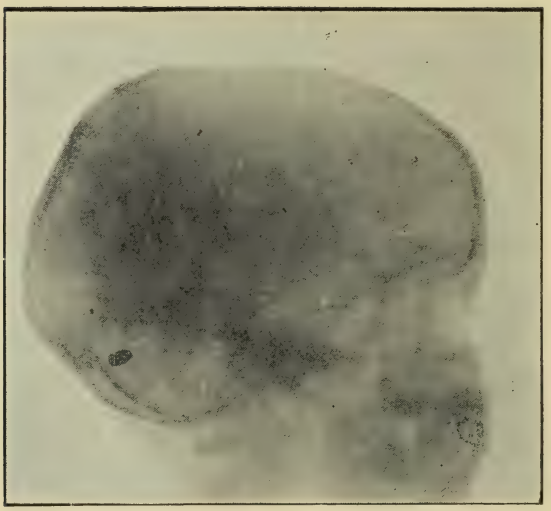


Fig. 3.—Bullet retained in brain; no operation; recovery.

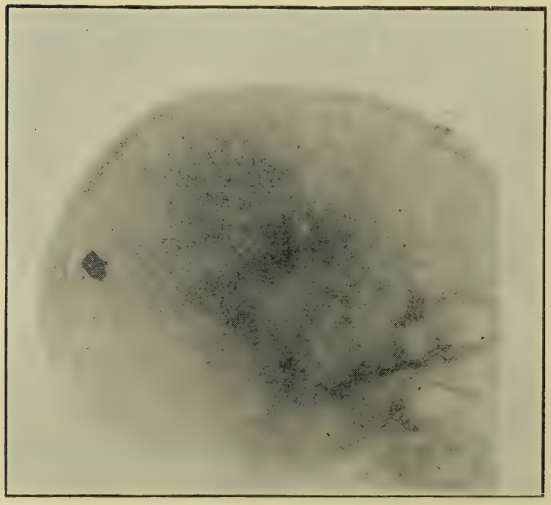


Fig. 4.—Bullet in cerebellum; paralysis of arm and leg; operation; bullet not removed; recovery.

Fig. 5.—Freeman. Gunshot wound of brain; paralysis of left leg; convulsions; operation; complete recovery.

Fig. 6.—Freeman. Anteroposterior view; bullet not removed.

Fig. 7 —Wakefield. Bullet retained in center of brain for three years; no operation; no sequelæ.

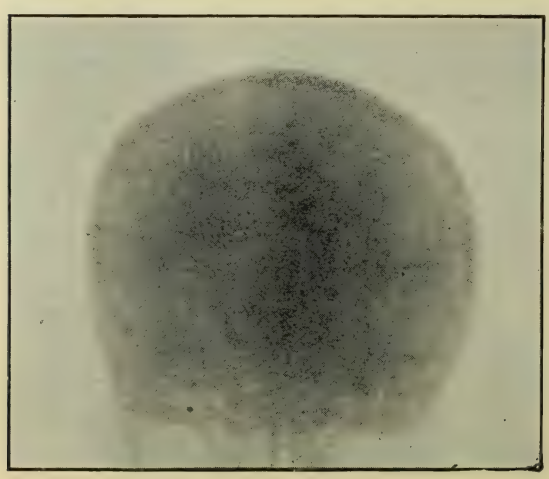


Fig. 8.—Wakefield. Anteroposterior view.

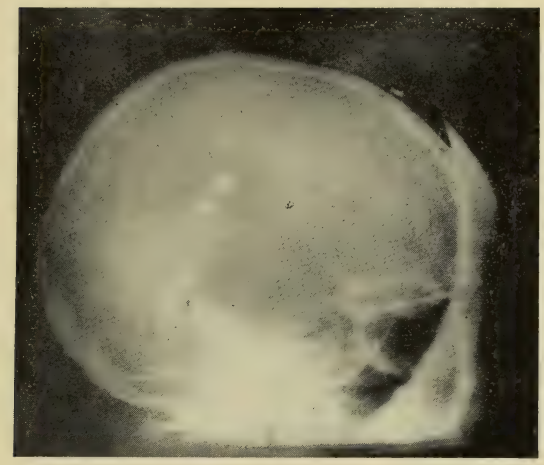


Fig. 9.—Wakefield. Side view. Picture made by superimposing a plate taken in 1917 upon one made in 1914 shows the bullet in practically the same position. The slight difference can be explained by the different angles at which the plates were taken.

Fig. 10.—Cerebral hernia. One of Sir Rutherford Morison's cases cured by "bipp." The picture was taken from the British Journal of Surgery.

Fig. 11.—Mooney. Cerebral abscess followed by cerebral hernia, abscess perforated skull; trephined; recovery.

Fig. 12.—Mooney. Photograph after recovery from cerebral hernia.

$2\frac{1}{2}$ inches just above the lateral sinus. Found extradural clot 2 inches in diameter by $\frac{1}{4}$ inch thick beneath fracture.

Result: Prompt recovery, union by first intention. Disappearance of the headache and return to entire consciousness followed directly on waking from the anesthesia.

Gutter Fractures

In gutter or tangential injuries the inner plate is always fractured; usually there are spicules of bone piercing and lacerating the brain, with intracranial hemorrhage. This type of trauma carries a high mortality. Mueller says that even with primary operation the mortality is 22.7 per cent.

Depressed fractures from direct localized blows, as from shell or shrapnel, are characterized by a high degree of concussion, fragmentation of the bone, laceration of brain tissue by bone or foreign bodies being driven into it, hemorrhage, etc. War injuries of this class correspond to those we are accustomed to see from automobile accidents, falls upon the head, blows from clubs, brick-bats, beer bottles, hammers and implements of various kinds. The following case is illustrative:

Compound Comminuted Fracture of Skull

Patient: C. Childers, N. Wilkesboro, N. C., aged forty-eight years.

Trauma: While partially intoxicated was struck on right side of head with quart bottle July 8, 1911.

Symptoms: Shock, concussion, semicoma, slow pulse and disturbed respiration.

Transportation: Was moved 100 miles to St. Leo's Hospital.

Examination: On entering hospital shock and concussion were less. Patient still semicomatose. Had urinary retention. Left leg and arm completely paralyzed. Right side normal. Head: On the right side of head there was a lacer-

ated wound of scalp and a large comminuted fracture of skull with numerous pieces of bone driven into the brain.

Operation: Craniotomy July 12, 1911, four days after injury. Semicircular scalp incision, depressed bone elevated, edges rongeured. Large extra- and intradural clots were found. Rubber tissue drainage.

Results: Wound healed by first intention throughout. Paralysis of leg and arm disappeared within a few days. No untoward sequelæ.

In the prevailing style of "over-the-top" fighting many punctured wounds of the brain are necessarily made. The following case is a good illustration of a punctured wound of the brain:

Fracture of Skull—Cerebral Abscess (*see Fig.* 2)

Patient: John Goode, convict, aged twenty-two years.

Trauma: Patient struck on head with sharp-pointed pick March 26, 1916.

Symptoms: Semicoma, headache, sepsis, no paralysis or convulsion.

Wound: One inch in diameter, situated behind center and to right of midline, infected and ranged. Wound in brain $2\frac{1}{2}$ inches deep filled with pieces of bone, wool, débris and pus.

Operation: April 3, 1916, eight days after injury. Wound cleansed, scalp edges trimmed. Brain wound cleaned out. Tube drainage.

Results: Uneventful recovery. Wound healed by granulation. No untoward sequelæ.

Penetrating wounds of the brain by the modern army rifle are destructive to a supreme degree. Bergman's experiments show that at fifty yards the bone and scalp are literally torn to pieces and the brain disorganized. At a distance of one mile the bullet penetrates through and through and not until the distance is over $1\frac{1}{2}$ miles does the bullet fail to emerge

after having entered the skull. However, the effects of bullets are modified by circumstances.

I have records of a number of patients with bullets and bone lodged in the brain. Indeed, in some the missiles have been retained for long periods.

Bullet Retained in Brain (*see Fig.* 3)

Patient: Taft Phillips, Denim, N. C., aged five years.

Trauma: Pistol bullet entered forehead above left eyebrow October 8, 1914.

Symptoms: Profound shock, concussion, irregular respiration, slow pulse. Patient came near dying while picture was being taken. No paralysis developed.

Treatment: After removal to hospital symptoms improved rather rapidly. No operation done.

X-rays: Show bullet lodged in posterior portion of brain.

Result: Prompt recovery. No untoward sequelæ.

Pistol-shot Wound of Brain—Bullet Retained (*see Fig.* 4)

Patient: H. Caviness, Carthage, N. C., aged six years.

Trauma: Accidentally shot just above midforehead.

Symptoms: Shock, concussion, coma, P. 80, R. 34. Six hours later shock and coma less. Paralysis of left leg and left arm.

X-rays: Show bullet in posterior fossa of the brain.

Operation: Wesley Long Hospital, August 20, 1917, eight hours after injury. Semicircular incision and trephined near wound.

Rongeured from trephine opening into wound.

Removed depressed pieces of bone, including an inch of the longitudinal sinus groove.

Cleansed and drained wound with tube.

Results: Operation wound healed by first intention.

Pistol wound quickly healed by granulation.

Paralysis of arm better next day; disappeared by third day.

Paralysis of leg better second day; disappeared by the fifth day.

Gunshot Wound of Brain—Late Operation—Bullet Retained (see Figs. 5 and 6)

Patient: Gooley Freeman, Greensboro, N. C., aged thirty years.

Trauma: Wife shot patient through head with pistol.

Symptoms: Coma. Semicoma, which increased to general coma.

Paralysis: Complete paralysis of entire left leg.

Wound: Left parietal, close to midline and toward front; wound infected and much depressed bone.

Convulsions: Frequent convulsions developed on seventh day and continued.

X-rays: Show bullet lodged in posterior portion right lobe of cerebrum.

Operation: September 4, 1915, eighth day, soon as patient was seen and examined. Trephined on right over left leg center.

Removed comminuted bone and cleansed wound.

Drained; no attempt made to remove bullet.

Result: Operation wound healed by granulation. Convulsions and coma quickly disappeared. Paralysis gradually and completely disappeared.

An interesting question in connection with retained missiles in the brain is whether or not they remain stationary. In the next case the bullet has remained *in situ* for three years:

Retained Bullet in Brain (see Figs. 7, 8, 9)

Patient: Thomas Wakefield, Greensboro, N. C., aged twelve years.

Trauma: Gunshot wound of head.

Symptoms: Patient fell but recovered consciousness in a very short while. No paralysis.

X-rays: Show part of split bullet under scalp; remainder in center of brain.

Treatment: Wound dressed; patient kept under observation. No operation.

Result: Patient recovered promptly.

Three years later: No neuroses have developed. *X*-rays show bullet in same position in 1914–1917.

The results of brain trauma are emphatic and worthy of our consideration.

Results of Brain Trauma

Death immediate or within a few hours.

Shock, concussion and compression.

Increased intracranial pressure due to:

- Shock.
- Hemorrhage.
- Edema.
- Infection.

Infection due to:

- Meningitis.
- Ependymitis.
- Cerebritis.

Cerebral hernia or fungus.

Death: This is where the grim reaper collects his greatest toll and robs both surgeon and patient of a chance. According to Fischer's statistics from the German Army during the Franco-Prussian War, "45 per cent. of 8132 gunshot injuries resulted in immediate death, and nearly one-half of those found dead on the field of battle had wounds of the skull."

With shock, concussion and compression we are all familiar.

Increased intracranial pressure is not so well understood. It is one of the three great destructive forces in brain traumas.

It is due to several factors: (*a*) shock, by which is meant a general disturbance of the brain; (*b*) hemorrhage, which if not sufficient to destroy life at once increases the pressure until the vital centers are disturbed; (*c*) edema, which always develops and greatly augments intracranial pressure.

There are many evil results of brain trauma, but the greatest of these is infection. It is the synonym of destruction, the destroyer of life itself. All war wounds are infected. The French speak of the first six hours as the period of "contamination," after that "infection." Infection takes three definite forms, each shading into the other. Strange as it may seem, meningitis *per se* is not so common or fatal a complication of brain trauma as is generally supposed. If the type of infection and the condition of the wound allow adhesions to form between the dura and subdural tissues, meningitis spreading from the wound or the so-called vertical meningitis is rather rare. If, however, the infection is of the streptococcic type, adhesions do not form and fatal meningitis almost always develops. Should infection invade the ventricles all authorities agree that the resulting ependymitis practically dooms the patient.

The most common form of infection is a more or less general inflammation of the brain substance itself. Infection greatly augments intracranial pressure, which in turn is the chief factor in the development of cerebral hernia.

Protrusion of the normal brain does not occur even although the dura be incised. In trauma the brain substance is forced through the skull opening until the equilibrium of intracranial pressure is restored. While the largest head clinic in this country never had a cerebral hernia greatly to its credit, it is one of the most common results of military brain traumas. Out of 610 head cases invalided back to England, 120 had cerebral hernia.

A cerebral hernia usually becomes profoundly infected, develops ulcers, granulation tissue, exudes foul discharges and often leaks ventricular fluid. Under these circumstances

it is called cerebral fungus. Cerebral fungus kicks back, as it were; it increased the intracranial pressure, which in turn causes more brain substance to extrude. Often it becomes strangulated and pulsation in the herniated part ceases (see Fig. 10).

Cerebral Abscess—Brain Fungus (*see Figs.* 11 *and* 12).

Patient: M. Mooney, cotton mill operative, aged thirteen years.

Trauma: March 10, 1913, patient struck right side of head, just above ear, against spinning frame. No scalp wound.

Symptoms: Continued at work, only slight pain. After three or four days developed much pain, fever, edema of soft parts and sepsis.

Operation: Saw patient April 17, 1913. Incision and drainage of zygomatic region. Considerable pus.

Result: Marked improvement.

Note: On May 19, 1913, patient returned to hospital; condition serious. Made free incision in temporal region. Found pus oozing through pencil-sized perforation in skull. Trephined. Removed abscess size of guinea egg, with well-formed sac.

Complications: Sepsis gradually subsided. Cerebral hernia (fungus) developed, discharging much serum.

Results: After trimming off hernia several times and treating parts antiseptically patient made a slow but perfect recovery.

Remarks: Evidently linear fracture, cerebral hemorrhage, infection through blood stream, perforation through skull, cerebral hernia.

I shall not go into detail regarding infectious agents, but simply remind you that the fields of Flanders have been under intensive cultivation for centuries and the soil is impregnated with not only the ordinary pus-forming cocci

but with gas and tetanus bacilli. For this reason a wound of the brain, indeed of any part of the body, is a more serious matter in Flanders than it would ordinarily be in America.

TREATMENT OF BRAIN TRAUMAS

Primary cleansing of the wound.
Removal to hospital for operation.
Taking of *x*-ray pictures.
Excision of scalp and bone wounds.
Discriminating removal of foreign bodies.
Covering of the exposed brain.
Complete or partial closure of the wound.
Only superficial drains, except in abscess.
Lumbar puncture, repeated as indicated.
Prolonged rest in bed.

This rapid survey of conditions found in brain trauma leads irresistibly to the conviction that the three great things with which the brain surgeon has to contend are: (1) increased intracranial pressure; (2) infection; and (3) cerebral hernia. Almost every other consideration is subordinate to these.

Bowlby and Wallace sum up the treatment of brain traumas quite forcefully. They say there are two reasons for head cases doing badly: (1) want of a good operation; (2) early evacuation of cases well operated upon. It seems that head cases stand transportation very poorly after they are operated upon. Therefore, it is far better to transport the head cases at once to the hospital, whether near or far, where the patient can remain for a long period. It has been found, in fact, that patients who were transported to the base hospital did much better than those operated upon close to the firing line under unfavorable environment. Neither a slow pulse nor depressed fragments of bone constitute an indication for immediate operation. The slow pulse rather suggests that an operation is worth while. The depressed

fragments as well as missiles had better be removed in a thoroughly equipped operating room by a skilful surgeon. And yet brain traumas should be operated upon at the earliest moment, day or night, that they can be gotten into a favorable environment.

Highly specialized surgery calls for specially qualified surgeons. Captain Bagley says: "We are confronted with the fact that there are very few neurological surgeons." Surgically speaking, "The field is ripe unto the harvest but the laborers are few."

To overcome this the Surgeon-General has provided for intensive training of a large number of general surgeons who already possess ample experience and excellent judgment and are willing to undertake the special work to fit themselves for doing brain surgery.

It is planned to expand the corps of brain specialists at least to 250. For this purpose the larger hospitals and clinics have placed themselves at the service of the Surgeon-General. Schools of brain surgery are being established at the University of Pennsylvania under the direction of Dr. Chas. H. Frazier; Chicago under Dr. Dean Lewis; in St. Louis under Dr. Ernest Sachs; and another in New York under Dr. Chas. A. Elsberg. The course is arranged to deal particularly with the fundamental subjects, anatomy, physiology and symptomatology of the nervous system. After receiving this intensive fundamental training, surgeons will be given the benefit of special clinical training in Europe and experience in a school for neurological surgeons which is being established in France within the war zone. What more splendid opportunity could one wish for training and experience in brain surgery? Those of you who have heretofore taken post-graduate courses have done so at your own expense. The Surgeon-General offers to give you the finest course in brain surgery, both theoretical and practical, that was ever conceived by mind of mortal man and to pay you while you are taking it. And the educational feature is the

best part of it when we remember that it also gives you a world-wide opportunity to serve humanity.

Does anyone hesitate because of the dangers incident to ocean travel or the firing line? Knowing the profession of the South as I do, I am confident of a ready response to this call for patriotic service.

Let me tell you of the spirit in which North Carolina responded to the appeal of the Surgeon-General for enlistment in the Medical Reserve Corps. She is one of the few States that quickly furnished her quota and that, too, without aid from itinerary boards or other agencies than the work of the State Committee of National Defense, Medical Section, and a few most excellent special examiners appointed by the Committee.

Some time ago I went to Raleigh to deliver a patriotic address before a body of medical men. At its conclusion Dr. R. H. Lewis, who is one of the Nestors of the profession in North Carolina, arose and told this narrative:

"Off the coast of North Carolina are the Diamond Shoals. They extend many miles along the coast and far out to sea. These shoals are so dangerous, so many ships have been wrecked here and thousands of lives lost, that the place is called by sailors the 'Graveyard of the Atlantic.' The Government maintains a light-house and a life-saving station at this point. A few years ago a terrific hurricane swept up the Atlantic Coast, spreading death and destruction in its wake. Washington sent out signals of the coming storm and ships scurried to safety while coastwise towns fastened down everything movable. As the storm moved northward it increased in velocity and destructiveness. When it reached Diamond Shoals it seemed to linger for a while as if to rebaptize the cemetery of its own making with the horror of its fury, as it had done a thousand times before.

"The inevitable happened. When the morning came a ship was seen stranded far out upon the point of Diamond Shoals. Captain Pat Ethridge, of the life-saving station,

called to his men to get out the life-boat. Accustomed as they were to obey orders, and fearing neither the ocean nor the storm, they responded with alacrity. But as they felt the force of the wind and saw the billows roll mountain-high and dash themselves upon shoal and shore they hesitated. They said: 'Captain, it is possible that we can reach the ship, but it is certain we can never come back.' Captain Ethridge looked again through his glass, saw the ship breaking up and the crew hanging as by a slender thread. Turning to his crew he commanded: 'Man the boat, men; we've got to go to that ship. We don't have to come back.' "

Following this story the President of the Society, a fine, stalwart fellow in the strength of mature young manhood, stood up, dressed in the uniform and insignia of a First Lieutenant in the Medical Reserve Corps of the United States Army, with his commission, signed by the President of the United States, in his pocket, with a seriousness of manner that indicated he was not unmindful of the wife and children at home, and said: "Gentlemen, I hold in my hand a telegram ordering me to the front. I have heard the call of my country. I shall go; I do not have to come back."

As I sat there I thanked God from the bottom of my heart that I, unworthy as I am, belong to a State, a nation, a profession that produces men of the type of Captain Pat Ethridge and that heroic doctor.

I am pleased to add that Captain Ethridge reached the ship, rescued every man on it and landed them safe on shore.

England has as her emblem the rose; France chose the lily; Scotland the thistle; Ireland the shamrock; Italy the leaves of the sturdy oak; the United States of America selected the stars.

All these beautiful emblems save ours are fruits of the summer. May God grant to our beloved Allies perpetual summer until Prussianism has been dethroned; until German autocracy, with all its inhuman barbarism, its outrage of chastity, its slaughter of the innocents, its hollow mockery

called "kultur," shall be stamped from off the face of the earth; until democracy, with all the glorious privileges that go with government by the governed, shall be enthroned throughout the world wherever the sun doth shine.

But, regret it as we most assuredly do, the winter of adversity may come upon even those we love best; the rose may shed its petals, the glorious lily may lose its fragrance, the thistle may decay, the oak leaf will feel the touch of frost, the shamrock may be Sinn-Feinned; but the stars—the stars shall shine on forever!

TERATOMA OF THE SACRUM; GLIOMA OF THE UPPER ABDOMINAL CAVITY: REPORT OF CASES

By James E. Thompson, M.B., B.S. (Lond.), F.R.C.S. (Eng.), F.A.C.S.
Galveston, Texas

It appears to me that neoplasms should be studied from three distinct stand-points: the anatomical, histological and histogenetic. The anatomical appeals most to the practical surgeon because it enables him to remove the growths safely and radically, and in so doing to satisfy both the immediate needs of the patient and his own standards of good work. Histological study follows closely on this, so closely, in fact, that in some clinics a histological diagnosis is given to the surgeon before the operation is completed and if the case demands it, before complete removal of a tumor is attempted. Histogenetic study, *i. e.*, the study of the origin of the cell of the tumor, is usually relegated to the pathologist, receiving, I am sorry to say, but scant attention from the surgeon as being a matter of mere didactic interest, with little practical bearing. Such a mental attitude is to be deplored, because it shuts the surgeon completely out of the romantic atmosphere of his specialty and reduces him to a mere materialist. The reproach has often been hurled at surgeons that they were degenerating into clever craftsmen, doing what they were told to do by the internists without personal knowledge of the reasons for operating. In some degree this reproach has been merited. The surgeons we revere most like John Hunter and Billroth were consummate pathologists.

If any apology is needed for the report of the two following cases it lies in the hope of placing before you in as simple a manner as possible the etiology of the reported tumors from an embryological stand-point.

Case I.—A female white baby, aged six months, came under my care suffering from a large tumor attached to the lower end of the sacrum and coccyx. At birth the tumor was about the size of an orange. The baby was well nourished and continued to grow normally and steadily without any interference with the natural bowel movements. The tumor seemed to increase in size proportionately with the growth of the child, until the child reached the age of five months, when the parents became alarmed by the rapid growth of the tumor. When the case came under my observation the tumor was as large as a cocoanut. It occupied the region between the lower end of the sacrum and the anal orifice, which latter was somewhat prominent and distorted. The growth was globular in shape, smooth in outline, elastic to the feel, and of homogeneous consistence. No cysts could be discovered. The skin was freely movable over its surface. It was firmly fixed to the sacrum and coccyx. Digital examination of the rectum revealed nothing abnormal. The mucous membrane was intact and free from signs of infiltration. It could not be moved freely from the growth. X-ray examination was negative as to the presence of bony or calcareous masses. A diagnosis of congenital sacral tumor was made. Operation was performed on February 11, 1915, under ether anesthesia. A transverse incision was made over the middle of the growth and the skin retracted upward and downward. The dissection was comparatively easy. The lower end of the sacrum was divided and the growth enucleated from above downward. In this way the recum was exposed high up as in a Kraske operation and the deep dissection carried out under the eye. The rectum was not seriously hurt, only a trifling amount of muscular tissue being removed with the growth. The sphincter muscles were undisturbed. Convalescence was

ideal, being unattended by any disturbance of the rectal functions. At the present time of writing (December, 1917) the child is in perfect health.

Description of Tumor. The tumor was as large as a cocoanut and showed the lower part of the sacrum and coccyx spread on its posterior wall. On section it appeared for the most part of a yellowish-white color and of a firm, homogeneous consistence. It looked like dense fat. Numerous serial sections were made which for the most part presented the appearance described below. In addition a number of islands of cartilage were found, one of them being of considerable size; also a few smaller islands of irregular shape and of a grayish-pink color, which strongly resembled the tissue of the brain; and also a number of cystic cavities, one of which was of considerable size.

The microscopic features are shown in the following series of photomicrographs for which and the attached legends I am indebted to my friend and colleague, Dr. V. H. Keiller.

The case conforms to the type of congenital sacral tumors that have been hitherto described,[1] *i. e.*, it contains elements of all of the three embryonic layers (epiblast; neuroglia; hypoblast; glands: mesoblast; fat, fibrous tissue and cartilage).

The exact origin of these growths has never been settled definitely. The theory usually advanced is that they are derived from the vestiges of the neuro-anal canal and the postanal gut. Another theory, viz., that they are the result of fetal inclusion, has little to recommend it, because there is no reason why fetal inclusion should occur in this particular part of the body.

In all probability the real explanation is to be found in unusual and misplaced activity of the posterior growing point of the embryo. This theory is defended strongly by Adami, whose able arguments and clear reasoning appear to

[1] Schwalbe: Morphologie der Missbildungen.

settle the question conclusively. The following description is taken from his text.[1] It is based on our knowledge of the growth of a plant: "If we consider that the first cell divides into two and then into four and each of the four again into two, we find that the two poles tend to become separated one from the other and each pole is advanced by the cells that are built in behind it. There are in the plant, from an early stage, two primary growing points, the superior forming the stalk and the inferior the root; the entire plant arises from one or other of these 'points' and the growing point is the most advanced part of the plant; the plant increases by growth backward and not forward from the growing point whose position is thus fixed at its apex.

"The growing point does not grow forward but is projected forward by the intercallation of the daughter cells behind it and the daughter cells are able to divide in a transverse way and give rise to special tissue cells. Secondary growing points can subsequently arise from the daughter cells, but their vegetative function must be exercised now in a direction more or less lateral and no longer axial. Similarly in animals there are two 'growing points:' the superior growing point is represented in the adult mammal by tissue about the situation of the pituitary body and the inferior by tissue somewhere in the sacral region. These two points lie beyond the extremities of the chorda dorsalis, an organ whose prominence in the early embryo and whose apparent unimportance in the formed fetus suggest that its function may be partly the keeping of the growth axially as a scaffolding might do in the earliest stages. These two growing points from the very origin of the embryo are being projected apart and the successive daughter cells given off from these give origin to the different organs of the body and even form secondary growing points for the limbs. . . . But there is a fundamental difference to be observed. Whereas in the plant

[1] Adami: Text-book of Pathology, p. 815.

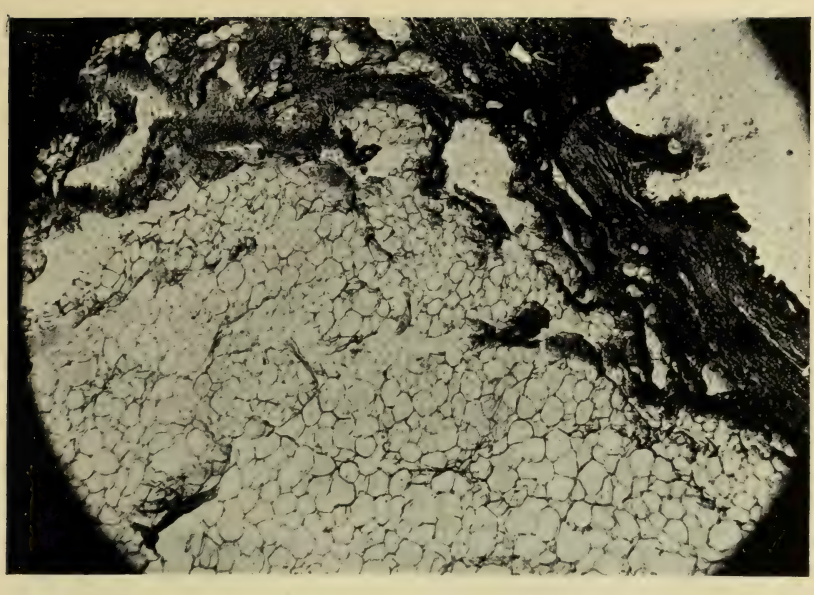


Fig. 1.—Shows fat composing the bulk of the tumor like normal fat in appearance microscopically, but grossly very light in color. At one corner of the section is seen the wall of a cyst with epithelial lining. Later figures will show this cyst wall more perfectly.

Fig. 2.—Shows an area of fibrous tissue, embryonic in type, infiltrating the fat. Grossly this is white fibrous tissue similar to that in the fibroma molle found in the uterus. This is slightly softer and more cellular than adult fibrous tissue. Mixed with it are some areas of fatty tissue.

Fig. 3.—This figure shows cartilage, embryonal in type, similar to adult hyaline cartilage but containing more cells. This and the two preceding figures are of mesodermal origin.

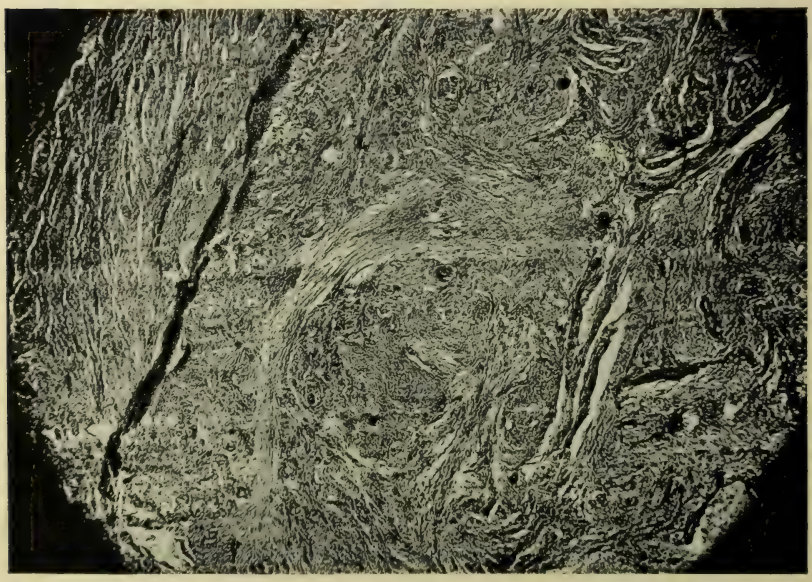
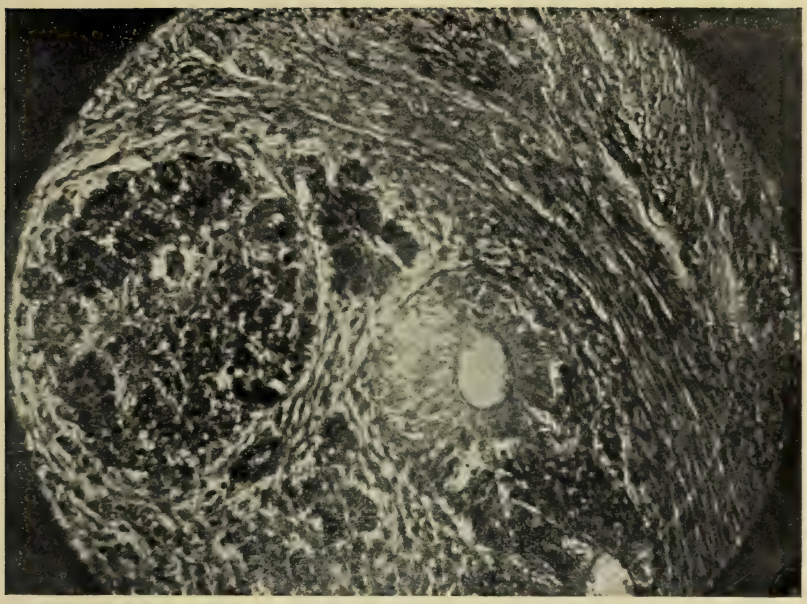
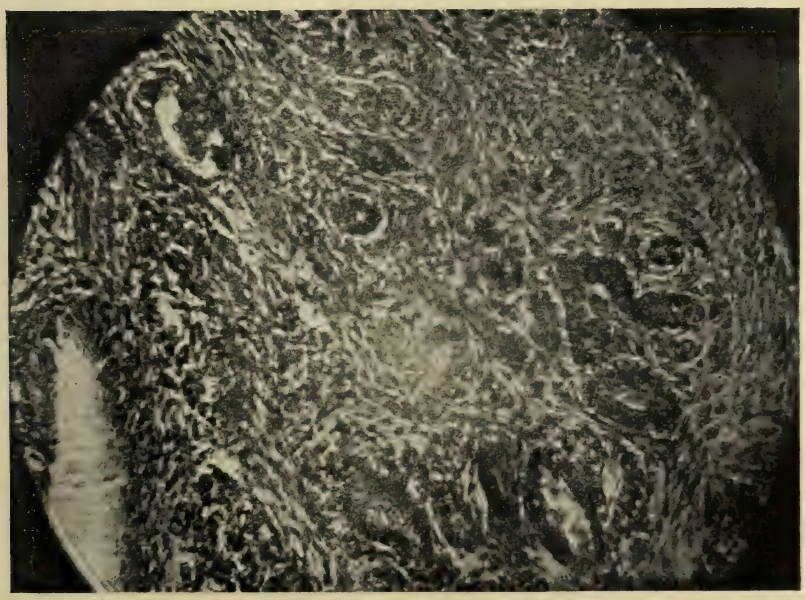


Fig. 4.—This section is taken from one of several small irregular areas, grayish pink in color, imbedded in the fat of the tumor. One or two of these contain small cystic cavities. Microscopically, as will be seen from the section, these areas resemble neuroglia. In the gross specimen, also, their color and appearance is very suggestive of nervous rather than of fibrous tissue.





Figs. 5 and 6.—Higher power pictures of the same area. In these some of the cells have a definite ependymal character, being arranged in a radial manner. Only a neuroglia stain would differentiate these areas positively from gland tissue; but the conjunction of cells arranged in this way with tissue so much like gliomatous tissue is fairly definite evidence of their glial character, and if glial, they are of ectodermal origin.

Fig. 7.—This shows the wall of a papilliferous cyst imbedded in the fatty material, containing a thick, tenacious, clear mucus, like normal mucus and not like the mucoid or gelatinous material usually found in tumor cysts. This tissue is suggestive of gastro-intestinal mucosa and therefore seems to be hypoblastic in origin. Unfortunately, mesoblast also gives rise to mucous glands (uterus, *e. g.*). The lining epithelium is simple columnar with goblet cells and simple tubular glands opening into the cyst. Deep in the tissue, it is difficult to tell whether the radial collections of cells are glandular or ependymal; the differential neuroglia stain is very uncertain in its results and an attempt made upon this was a failure—failing, also, in spinal cord—so that it proved nothing. In places the epithelium lining the cyst wall is stratified quamous.

Fig. 8.—This shows another section of a cyst wall, a part of the same cyst in which the deeper layers are more definitely fibrous. The papilliferous arrangement is very accentuated.

Fig. 9.—Shows a section taken through the wall of one of the blood clots in the tumor. It contains a portion of the clot. It will be seen that there is no epithelial lining to the blood space, nor is there any epithelium lining other cyst-like cavities containing gelatinous substance which are found elsewhere in the tumor. The lighter portion of the slide shows collections of fine, faintly pink stained fibrillæ which I interpret as neuroglia fibrils, since they do not take a connective-tissue stain with Van Giesen's fluid. The darker portions represent collections of glial cells.

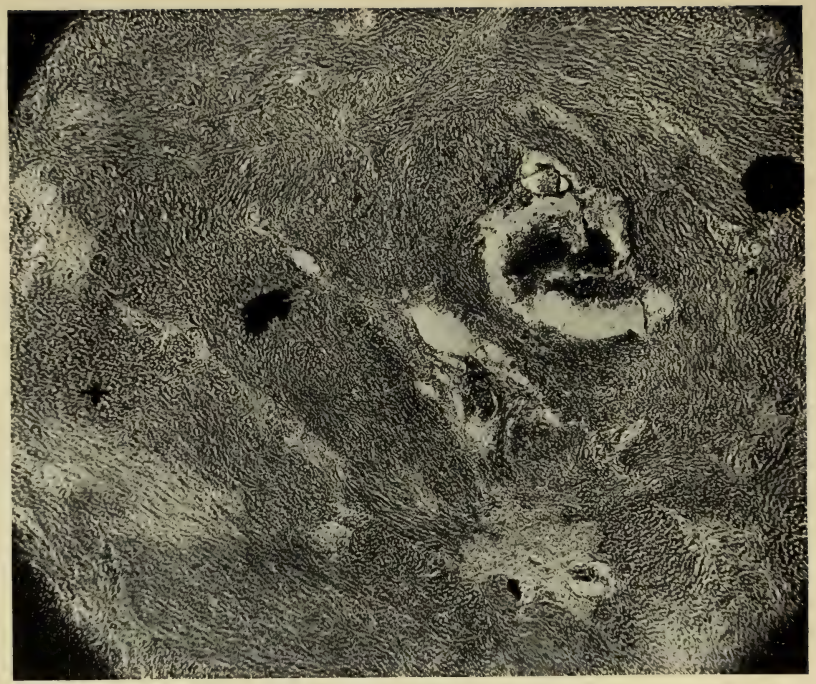


Fig. 10.—Shows another area of tissue containing one small hemorrhage about the middle of the slide and several of the lightly stained collections of neuroglia fibrils. In the darker, more cellular portion of the section some of the cells have a radial arrangement, suggestive of ependymal cells. Such an area, marked with a small cross, is shown in higher magnification in the following figure.

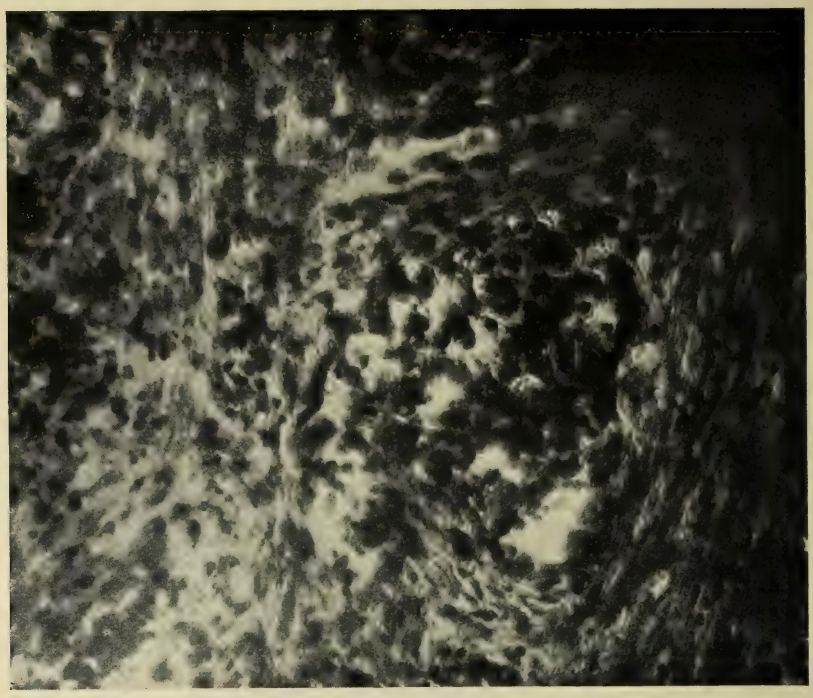


Fig. 11.—Higher power photograph of one of the radial collections of cells indicated in Fig. 10, and strongly suggestive of ependymal cells.

Fig. 12.—Another very low power photograph from the opposite end of the tumor, taken to emphasize the uniformity of its structure. Here again are light areas containing few cellular elements, and probably representing collections of neuroglia fibrils. In the more cellular portions are groups of round cells, some of them arranged in a radial manner, and many collections of spindle-shaped cells with indefinite outlines, of which higher power pictures follow.

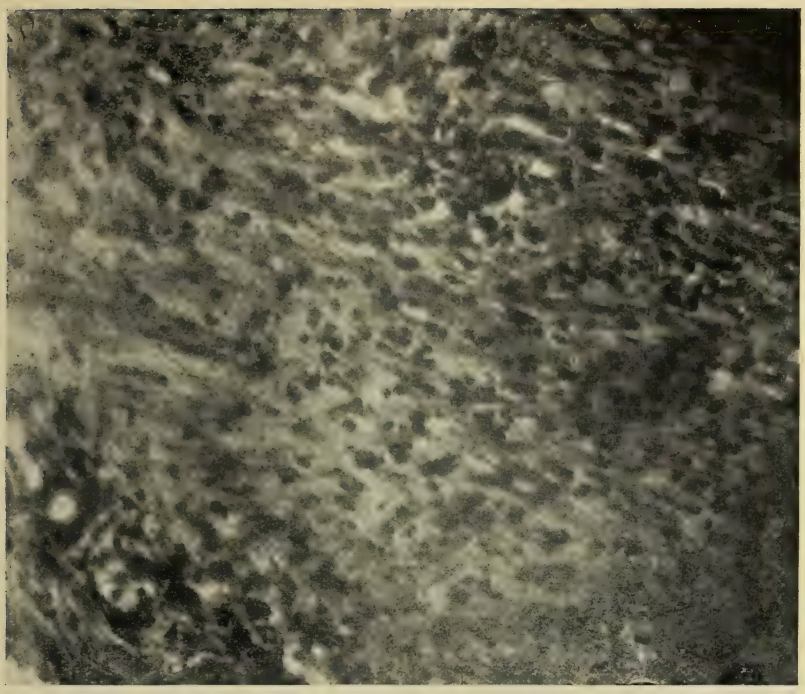


Fig. 13.—High power picture of a portion of the section just shown. These areas of round and spindle cells mixed together are very much like sarcoma and probably indicate sarcomatous change in the glioma.

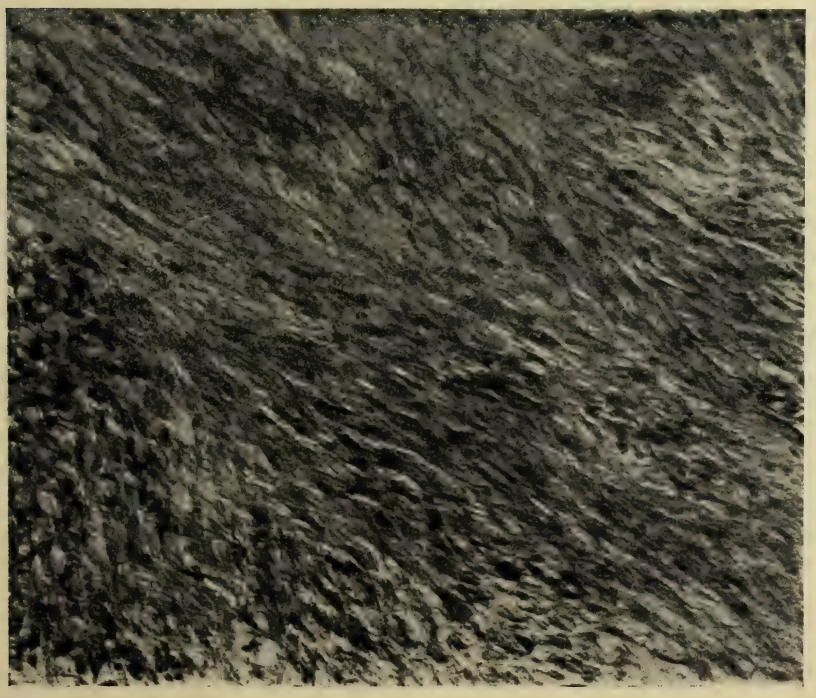


Fig. 14.—High power photograph of another area from Fig. 12, showing a collection of indefinite spindle cells, very suggestive of glial cells.

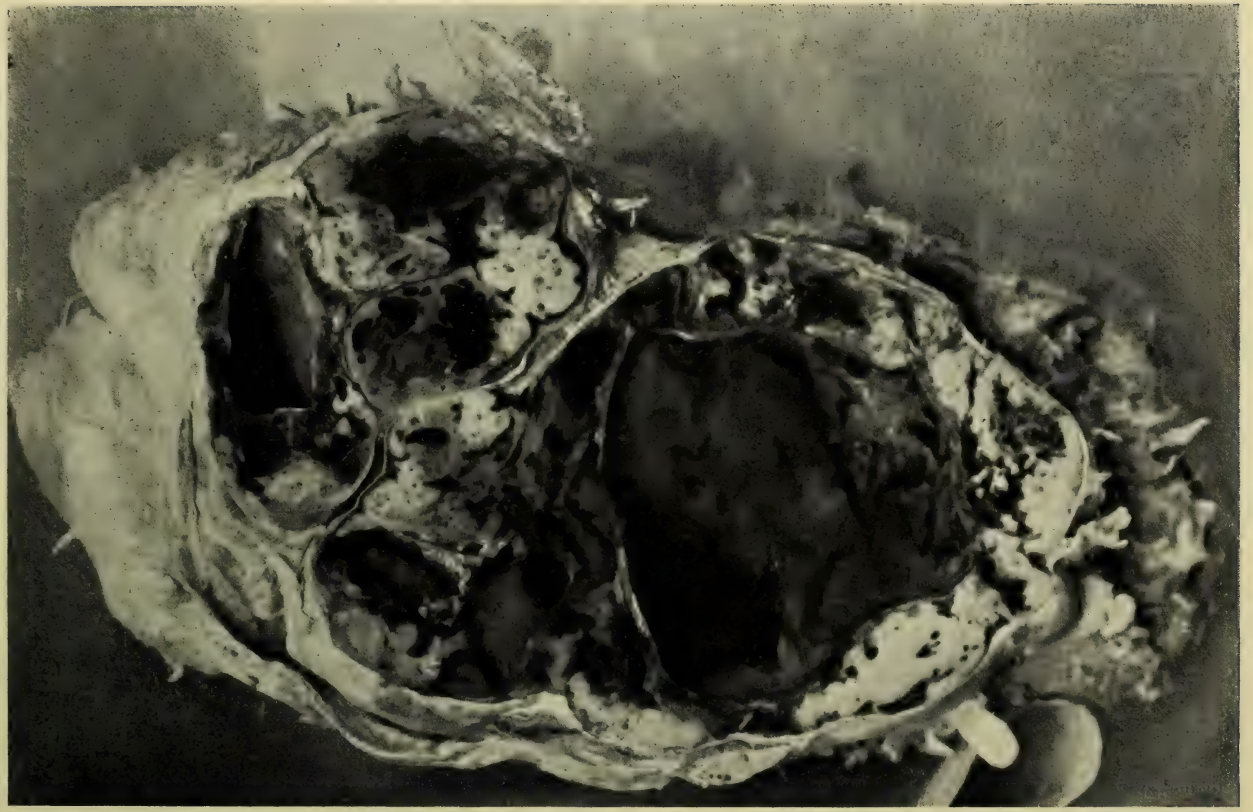


Fig. 15.—Section of the glioma of the upper abdominal region. The black areas represent blood cysts and blood extravasation. The white areas represent the intrinsic tissue of the tumor. Notice that it is mottled with small extravasations of blood.

the primary growing points are active during the whole of its existence, in the animal they cease to functionate as such when the analgen of the brain and the rest of the nervous system have been developed.

"The cells of the growing points consist of totipotent cells which are capable of giving rise to tissues of all orders belonging to the three primitive germinal layers. If, therefore, the growing points continue active large masses of such tissue may be formed at either end of the body. These masses may contain bone and soft tissues representative of meso-, epi- and hypoblast. Those occurring at the anterior end of the body arise from the base of the skull just behind the pituitary fossa. They are spoken of as epignathus. Those at the posterior end of the body are situated at the lower end of the sacrum and form the congenital sacral tumors."

Case II.—Glioma of the upper abdominal region. C. A., aged thirty-nine years, colored, was admitted to the John Sealy Hospital on November 21, 1917, suffering from a large tumor situated in the upper abdominal region.

History of the Disease. About four months ago he noticed that his stomach was becoming bigger. He was then a large man, weighing about 200 pounds, and as he suffered from no pain or other discomfort he thought he was getting fatter. The tumor grew rapidly and he lost about 50 pounds of weight in four months. He believed it showed itself first in the neighborhood of the umbilicus. There was no fever. It was his first sickness, so he did not seek advice, until the size of the tumor alarmed him. The day before admission to hospital he had slight pain over the tumor which spread to the pit of the stomach and over the left side of the chest.

Previous History. Gonorrhea at the age of twenty-one. No history of syphilis; had been an unusually strong and healthy man. No history of dyspepsia or any stomach trouble.

Present Condition. The patient looked fairly well nourished and weighed about 150 pounds. He complained

of nothing but the inconvenience of the abdominal tumor. It was not painful, nor was it tender on reasonable manipulation. The tumor was large. It reached from about two fingerbreadths below the umbilicus to the upper part of the epigastrium, the left side of which it filled. To the right it reached the middle of the rectus abdominis muscle. To the left it filled the upper abdominal space and occupied completely the left epigastrium and hypochondrium. The percussion note was dull over the whole area and no shades of difference could be detected in its various parts. It did not move appreciably with respiration. It seemed to be firmly fixed to the posterior abdominal wall. The surface was roughly nodular. There was no pulsation.

Urine Examination. Acid; sp. gr., 1012; sugar, negative; albumin, a distinct ring; no casts.

Blood Examination. Red cells, 3,944,000; white cells, 6200; hemoglobin, 80 per cent.; polymorphonuclears, 50 per cent.; lymphocytes, 49.5 per cent. Wassermann reaction, strongly positive.

No examination was made of the stomach contents.

Diagnosis. A tentative diagnosis of retroperitoneal sarcoma was made and an exploratory operation was performed on November 24. A large angular incision was made, the vertical cut passing downward through the inner edge of the left rectus from the epigastrium to the umbilicus, and the lateral cut from the lower end of the vertical upward and backward to the tip of the twelfth rib. This gave a full exposure. The growth was found free from attachments to the transverse colon or to the right half of the stomach. It seemed to have pushed its way forward between the greater curvature of the stomach and the transverse colon. It was very nodular and cystic. It was covered in front by a thin layer of omentum in which very large veins coursed. Followed to the left it fused with the spleen. To the right it was free. Posteriorly it could be lifted from the aorta. At first it looked an impossible task to remove it, but after division of the

omentum, which covered it in front and opening the lesser sac and lifting up the stomach, it was possible to separate it from all the anterior surface of the pancreas except about two inches of its tail. The left edge of the growth appeared to be jammed tightly between the tail of the pancreas and the spleen. The greater curvature of the stomach was also adherent to the growth over a small area. The tail of the pancreas and the splenic vessels were isolated and clamped, and a clamp was placed on the greater curvature of the stomach and the growth removed with remarkably slight hemorrhage. A careful examination of the other abdominal organs revealed no abnormality.

A drain was placed near the stump of the pancreas and the abdomen closed. Convalescence was unattended by any irritating discharges.

After-history. The patient returned to his work as a drayman six weeks after the operation, and since that time has worked every day without fatigue. On October 15, 1917, I examined him carefully. There was no symptom of recurrence. He was sound and robust and weighed 157 pounds. Blood count: Red cells, 3,944,000; white cells, 6200; polymorphonuclear neutrophiles, 50 per cent.; lymphocytes, 49.5 per cent.; transitional forms, 0.5 per cent.; hemoglobin, 80 per cent. Urine straw colored; acid; specific gravity, 1012; distinct ring of albumin; no sugar; a few leukocytes; amorphous urates; a few clear epithelial cells. Wassermann reaction, strongly positive, 4+.

Description of the Tumor. The specimen removed was as large as a Rugby football. It consisted of the tumor, of the tail of the pancreas, the spleen and a part of the greater curvature of the stomach. Both spleen and pancreas were tightly spread over the left edge of the tumor. Neither organ seemed to be infiltrated. The stomach wall was infiltrated and the growth projected slightly into the lumen. The tumor was very irregular in outline and its surface was nodular. Numerous blood cysts bulged from its surface. Its blood

supply appeared to be derived entirely from the splenic vessels.

Section of the tumor showed numerous cysts of a large size separated from one another by septa of considerable thickness. The cysts contained blood in various stages of disintegration. Its color was mainly of a deep maroon tint. There was no gross evidence of intracystic growths. The septa consisted for the most part of tissue of a yellowish-pink color and firm consistence, in which were found numerous areas of blood extravasation also of a deep maroon color. These septa evidently represented the main part of the original tumor. The gross features are well represented in the plate.

The microscopic characteristics are shown in the attached series of photomicrographs.

The pathological interpretation of the tumor is entirely our own, but we feel a little hesitation in insisting on our view obstinately. I have submitted the slides to my friend and former colleague, Dr. Allen J. Smith, of the University of Pennsylvania, whose opinion is that it probably resembles a chordoma which has undergone a sarcomatous change.

If the gliomatous nature of this tumor is admitted it probably takes its origin from one of the sympathetic plexuses of the upper abdominal cavity, in this particular case the plexus splenicus. Tumors of large size have been found arising in the upper abdominal cavity from these plexuses and from the adrenal gland which on microscopic examination have shown ganglion cells, axones and dendrites (Adami).

We have not been able to find indubitable evidence of ganglion cells, although in one specimen an area was found in which several bipolar cells strongly resembling ganglion cells were clearly distinguished.

CONFLICT OF CLINICAL WITH MICROSCOPIC EVIDENCE IN THE DIAGNOSIS OF TUBAL AND OVARIAN PREGNANCIES

By J. Wesley Bovée, M.D., F.A.C.S.
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To the careful observer the removal from the abdomen of a woman during the childbearing age of a tube or an ovary, plainly the source of a more or less copious hemorrhage, does not carry conviction that pregnancy in such structure was the prime cause. He demands more positive evidence of such ectopic pregnancy. If fetal structures are not promptly recognized by naked-eye appearances he seeks the aid of the microscope. He will find in a considerable proportion of such cases no microscopic evidence of gestation. The symptoms of the illness may be very suggestive of pregnancy quite frequently. Having found a rather surprising number of such cases in my work, and having seen many cases operated on in various clinics which, in my judgment, belonged to this class, and which were promptly and conclusively diagnosed as tubal pregnancy by the surgeon operating, I have been led to again call your attention to this matter. Some of our members may remember I presented this subject to this Association in St. Louis in 1897. But as that was twenty years ago, and a new generation is now with us, I hope this repetition may be pardoned, and, too, that occasion will not arise to present it twenty years hence.

The recognition of this type of hemorrhage in the non-pregnant state is not of recent date. Scanzoni reported

an autopsy he performed in the case of a young girl dying suddenly during menstruation. He found 3 liters of blood in the peritoneal cavity that had plainly escaped from a ruptured ovarian follicle. It is probable exhaustive means to exclude ovarian pregnancy in that case were employed, and I am inclined to believe the pathology present was similar to that of the many less severe cases we have encountered in recent years in which the absence of pregnancy was proved.

E. B. Cragin[3] was disappointed in 12 consecutive cases of supposed ectopic pregnancy that microscopic evidence did not confirm his diagnosis in 6 of them. He states these 6 had very good ectopic histories, but proved to be ovarian and tubal hemorrhages. From an observer as careful as Cragin this evidence is very striking. Doran[5] reported a case diagnosed as ruptured tubal pregnancy in which he found an *undilated* tube from the fimbriated end, from which a large amount of blood had escaped, forming a large pelvic hematocele. A microscopic examination of this tube showed no evidence of either pregnancy or inflammation. Edgar[6] reports one in which a supposed ectopic pregnancy proved to be a ruptured ovarian hematoma, with a resulting large pelvic hematocele. Hind[12] states that an emergency operation for ruptured tubal gestation was done and several pints of clots were turned out of the abdomen. The uterus and both tubes were normal, but that the left ovary was ruptured and oozing blood. The pathologist, Mr. Targett, found only an apoplectic Graafian follicle, with rupture of the ovary and no evidence of pregnancy. Bland-Sutton[22] and Galabin[9] stated twenty-five years ago that by no means must cases of hemorrhage in the tube be conclusively diagnosed as ectopic gestation. The literature teems with this variety of mistakes in diagnosis. But about 1890 the gynecological world was astounded by the epoch-making report of autopsies upon 35 women dying from ruptured ectopic pregnancy, made by H. F. Formad[8] to the Pathological

Society of Philadelphia. He had examined or witnessed autopsies on over 3800 women, and among them occurred these 35 cases of ectopic gestation. His analysis gave very impressive statistics. The women were all between the ages of twenty and forty years. "I may now state that I now class hematocele of the tubes as ectopic pregnancy even if no fetus is discovered," was one of his dicta. No case was beyond four months' development, not one extraperitoneal rupture of the sac was encountered; he seemed to have firmly concluded that all intraperitoneal ruptures of the gestation sac cause death and that those saved by operation were necessarily extraperitoneal. He gave the encouraging information to diagnosticians that the *uterus invariably contained a decidual membrane or very plain remnants of one* if previously expelled. The microscope confirmed this statement *in every instance.* All were of the working class or housewives in moderate circumstances, and at the time of the fatal seizure were usually working — washing, housecleaning, carpet shaking or lifting: 1 was ovarian, 3 interstitial and 31 tubal.

This report caused every surgeon and gynecologist to search "No Man's Land" for cases of ectopic pregnancy. Success attended their efforts to such an extent that, all praise to their zeal, rupture was discounted. Lawson Tait and his American pupil, Arthur W. Johnstone, as well as that sturdy old New Englander, S. C. Gordon, attacked these cases at the moment of discovery, as did Matthew D. Mann; others have since continued their work, and today no condition of ectopic pregnancy, except the extremely tragic ones, is considered too formidable for the only proper treatment, surgical. In the midst of this surgical industry was created the carelessness in diagnosis to such an extent that I feel justified in making this protest.

I have already mentioned the evidence of Cragin, Galabin and Bland-Sutton, and in 1904 I reported 10 cases I had encountered. J. Price,[17] H. P. Newman,[15] Griffith,[11] Croom,[4]

Ruge,[18] Goodell,[10] Morrison,[14] Doran, Edgar, Hind, Wilson[24] and a host of others have reported such instances of diagnostic error. Since the 10 cases referred to were reported I have had 19 cases, 15 of them being tubal and 4 ovarian, of hemorrhage from the ovary and tube that could not be diagnosed as ectopic pregnancy at the time of operation. In 3 of the tubal cases and 1 of the ovarian the microscopic examination reports have not been received from the laboratory. Of the tubal hemorrhages but 2 were decided to be from ectopic pregnancy. In 1 a uterine decidua without chorionic villi, together with marked tubal laceration, were the only criteria upon which we finally diagnosed tubal gestation. The other that of a single woman, aged thirty-seven years and never pregnant before, had suffered four months from almost continuous uterine bleeding, with pain in the right lower quadrant, which forced her at times to go to bed. The outer half of the right tube had a transverse diameter of about 4 cm., the fimbriated end being dilated. It contained a coagulum of blood attached to the tubal mucosa by a pedicle 3 cm. by 7 cm. The pathologist reported no fetus, but that the tube showed the presence of villi and decidual formation. It is interesting to note that this positive proof of tubal pregnancy was present four months after the beginning of the tubal abortion. It has been claimed such evidence disappears in much less time than four months. We have then 15 cases of tubal hemorrhage, in 3 of which microscopic evidence is not available. In the 12 cases upon which microscopic examination reports have been made, but 2 gave positive evidence of ectopic pregnancy. In the 4 of ovarian hemorrhage 3 furnished negative reports as to pregnancy and 1 has not been decided.

Ovarian and Tubal Hemorrhage. *Ovarian hemorrhage* may take place in a Graafian follicle or in the stroma.

Ovarian hematoma is a condition that was long since recognized. Puech, in 1858, declared ovarian apoplexy actually exists and may cause death; it may form a hematoma and

may or may not be absorbed. The condition has been so often encountered, as also have its dangerous associated conditions, that none should regard it as a myth or even a curiosity. Hemorrhage in the ovary occurs normally as it does in normal parturition. It may occur in Graafian follicles or in an ovarian cyst when the venous circulation of its pedicle is obstructed by twisting of or pressure exerted on that structure. It may occur in the stroma, constituting a true hematoma. It may occur in toxic conditions, causing several accumulations of variable size like those found in the kidney and other organs, and is noted in autopsies on patients dying of typhoid fever or other conditions having a local septic origin, such as puerperal sepsis or perforated gastric or duodenal ulcer (Wilson). Ovarian pregnancy is a rare cause. Hemorrhage into the stroma or into a follicle may occur in the newly born infant, fully developed, as in the case of Schultze;[20] in quite young girls at or near the first menstrual period and in early adult life or during the childbearing period, and may be accounted for in the non-infected cases by undue hyperemia, notably during the first few menstrual periods. Great sexual excitement is believed to be a quite common cause. Wilson states there is one case on record of a woman who died from shock on the eve of her marriage as the result of a profuse intraperitoneal hemorrhage from a ruptured corpus luteum. In early menstrual life profuse and prolonged uterine hemorrhage is far from uncommon, and it is urged by some writers that this is from the irritation caused by continued bleeding from the ruptured Graafian follicle. Probably you are all familiar with Scanzoni's case of a girl dying from such profuse follicular hemorrhage during menstruation. At autopsy he found 3 liters of blood in the peritoneal cavity. A considerable number of cases have appeared in literature, like that of Fordyce,[7] in which women, especially girls, exposed to cold and wet immediately preceding or very early in a menstrual period have been the victims of ovarian hemorrhage so great in amount as to

demand immediate abdominal section. In the inflammatory cases a quite well-defined sclerosis of the ovary has taken place, giving rise to excessive formation of connective tissue in the ovary, with fatty degeneration of the bloodvessels, which rupture easily. It is probable in such changed state that expulsion of the ovum is retarded and the consequent prolonged congestion provokes hemorrhage into both the follicle and the stroma. When rupture of the bleeding ovary occurs, whether naturally from a Graafian follicle or pathologically from overdistention from blood accumulated in its stroma, free exit is given to escaping blood and hemorrhage is encouraged, more especially during ovular hyperemia. The rapidity of outpouring of blood varies from a few drops to such large quantities as have been mentioned.

Schroeder[19] states that rupture of a Graafian follicle may in exceptional cases be followed by so profuse a hemorrhage that it can (1) prove fatal; (2) cause peritonitis which may terminate fatally; (3) produce a capsulated blood-clot in the peritoneal cavity; (4) produce a retro-uterine hematocele.

Tubal hemorrhage is not so well understood as is that from the ovary. While ectopic pregnancy is the active agent in its production in a preponderating proportion of cases, yet no one can doubt its occurrence quite frequently in post-infectional conditions, and in some instances reports have distinctly stated neither inflammation nor pregnancy was evident. In my judgment the cases in which local pregnancy, the result of infection or traumatism, are absent must be extremely rare. The relation of ovarian and tubal hemorrhage to intraperitoneal hematocele needs no elucidation from me.

Causes of Error in Preoperative Diagnosis. It is not difficult to understand how before operation the pelvic condition may erroneously be diagnosed as ectopic pregnancy in its early months. Nor can one blame the operator who diagnoses this condition, at operations, if he recovers a fetus or other fetal structures readily recognizable microscopically.

But to insist on such diagnosis at the time and later, in the absence of such evidence, is carelessness or worse. The aid of the microscope is absolutely necessary in this situation. The age of the patient, her record as to pregnancies and recent amenorrhea, the nature of the attack, particularly the character of its onset and its subsequent history as to shock, internal hemorrhage and metrostasis, each offers opportunity for error, inasmuch as they are so identical in both conditions in early cases or even those that have progressed for several weeks or a few months after the onset of attack.

Age. Ovarian hemorrhage has often been found in the newly born infant, but not later until the full development of the ovary has been accomplished, which is generally regarded as being indicated by the establishment of the menses. At this time the second age point of ovarian hemorrhage is reached and the confounding of this condition with ectopic pregnancy. The patient is now in her procreative period of life and may have either condition.

In ectopic pregnancy a history of sterility for several years is common. All of Formad's cases had previously aborted or borne children. The cases cited of Cragin, Doran, Fordyce, Hind, Lee,[13] Wilson, myself[2] and many others gave such history. As to amenorrhea, it seems to be as constant in the non-pregnant as in the pregnant types being considered. The ectopic gestation patient may have had a mild onset. Steffarson,[21] who studied 1000 cases of this form of pregnancy occurring between 1897 and 1907, states (l. c., p. 99): "The investigation of the histories of the ruptured cases shows, in the main, that primary rupture may occur with little or no pain." Many of the case reports of the non-pregnant cases under consideration mention the gentle onset. And, again, in either contrasted condition the onset may be very sudden and almost tragic from the suddenness and degree of the localized pain, shock, collapse, etc. Gaillard Thomas's[2] patient was stepping into a carriage when the sudden localized

pain and collapse occurred that was recognized as rupture of a tubal gestation. Edgar's patient, in whom he found a ruptured ovarian hematoma, was undressing to go to bed when her seizure was similar, and Hind's, of the same condition, was similarly attacked. In the slow cases—probably of the tubal pregnancy patients, the tubal abortions most commonly—the prolonged uterine bleeding is a feature of both the pregnant and non-pregnant conditions.

In fact, no man is wise enough, in my judgment, to distinguish before operation between early ectopic pregnancy and ovarian and tubal hemorrhage except he have special evidences of pregnancy, such as breast signs, gastric disturbances of short duration or knowledge of the uterine interior. And I need not attempt to instruct you in respect to those features. If upon operation no further evidence of ectopic pregnancy is found the diagnosis of that condition must be discarded.

To further illustrate the difficulty encountered in arriving at a diagnosis in some cases of ruptured tubal pregnancy, even when the history is quite clear, is presented in the following case:

Carrie T., aged twenty-three years; colored; married. Admitted to Columbia Hospital for Women June 10, 1910, complaining of pain in the lower right quadrant, epigastrium and head; had had one child and one miscarriage.

Menstrual History. First menses occurred at age of fifteen years; has been irregular, profuse and painful, usually lasting seven days; last period began about May 26. Last night was attacked with severe pain in lower part of abdomen, radiating to the back, nausea and vomiting. At seven that evening she felt a sudden, severe, colicky pain in the lower right quadrant followed shortly by vomiting. There was no bleeding at the time, but four days later, while in hospital, began to menstruate, the scant flow continuing for three days.

Operation, June 17. Double salpingo-oöphorectomy.

Both tubes found enlarged and adherent, neither containing fluid except as follows: one inch from cornu the right tube was flattened out into a ragged elliptical area about 1.5 by 2 cm., believed to be covered largely with mucosa. A large amount of old blood-clot, the largest being 5 to 7.5 cm. in diameter and near the cecum, was found in the pelvic portion of the peritoneal cavity and extending above the cecum.

The uterus was normal in size and in good position. The right appendage was referred to the hospital pathologist, the late Dr. John S. Neate, pathologist, Army Medical Museum, who reported as follows: "Specimen consists of one tube and an enlarged and distended ovary. The tube was enlarged, but not more than three-quarters of an inch in diameter, presenting a rupture at the juncture of the outer and middle third, about three-quarters of an inch long, with everted, thick edges. Numerous sections were taken by the class at the Museum through the rupture and the proximal and distal portions. No evidence of fetal tissue; practically no decidua. A small amount of purulent exudate in ruptured portions; the lumen is open on each side with little change in mucosa. Evidently one or more diverticula in the tube, since no contortion of tube would give the picture seen. Not entirely demonstrable, but clearly suggested that ovum must have reposed in pocket of diverticulum. Musculation of tube usually thick, ostium would have allowed of abortion if in proper lumen or in walls of tube. Inferred that ovum or fetus must have been a young one. Ovary shows beginning of adenomatous cysts, with marked sclerosis. In areas little or no ovarian tissue; very few diseased follicles. Noted that corpus luteum must have formed in opposite ovary. Specimen is peculiar in presenting so little evidence of ectopic pregnancy, apart from the ruptured tubal walls."

This case shows presumptive microscopic evidence of ruptured tubal gestation and microscopic and clinical pictures but little more positive.

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THE UTILITY OF END-TO-END ANASTOMOSIS BETWEEN SMALL AND LARGE INTESTINE

By D. C. Balfour, M.D.
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The restoration of the continuity of the small and large intestine following resection of the ileocolic coil is not the least important step in the operation. The exploitation of colonic resection for intestinal stasis has at least resulted in demonstrating the disadvantages of lateral anastomosis between the small and large intestine and in the rather general adoption of end-to-side union. The latter method has no seriously objectionable features, but it involves two steps which are essentially additional—namely, the closure and inversion of the end of the large bowel and the formation of a separate opening in it for the implantation of the end of the small intestine. Axial union in any part of the intestinal tract is manifestly superior to all other methods, provided the operation can be done with safety, with the preservation of good function and with the avoidance of late complications.

Recently, Lockhart-Mummery has exposed some of the fallacies in the attitude toward axial union of the large intestine showing that the relatively high mortality and morbidity from leakage are not due to inherent faults in the operation, and has described a method by which end-to-end anastomosis of the colon can be done with safety.

A more or less analogous situation exists in regard to end-to-end anastomosis of small and large intestine. It is quite

evident that axial union between ileum and colon is given little or no consideration in the literature and, in so far as we are aware, the procedure is not utilized to any extent among surgeons in this country. Our experience has shown that end-to-end anastomosis of ileum and colon can often be easily and safely performed, which leads us to believe that the general attitude toward the operation is unwarranted. It is for this reason that I bring the method to your attention.

It should first be said that we have thus far recognized the apparent limitation of axial union to those cases in which there is a dilatation of the small bowel, due to a chronic obstruction from some well-defined pathological condition such as cancer (Fig. 1) or hypertrophic tuberculosis. Under such circumstances the method has been exceedingly satisfactory. Resections of the ileocolic coil for conditions other than those associated with such definite pathological processes are relatively rare in our clinic, but when these other indications do arise and the ileum is small there seems to be no reason for abandoning the end-to-side anastomosis. At the same time, axial union has been so satisfactory in the group of cases in which the caliber of the small intestine more or less approximates that of the large intestine that it is quite possible by the employment of technical procedures (those of C. H. Mayo, for example) to increase the caliber of the smaller segment by cutting the bowel-end obliquely without sacrificing its lumen or splitting the bowel opposite its mesentery, the scope of axial union may be extended to include cases in which little or no dilatation of the small intestine has occurred (Fig. 2, *b*).

In carrying out end-to-end anastomosis between ileum and colon by suture we have followed in general the method used by Lockhart-Mummery in axial union of the large intestine, the anastomosis being made in the following manner: The resection having been completed the two stumps of the intestine are isolated by protective gauze pads, rubber-

FIG. 1.—Carcinoma of the ascending colon with dilated cecum, dilated ileum and contracted colon. Cuff of omentum dissected back, preliminary to resection.

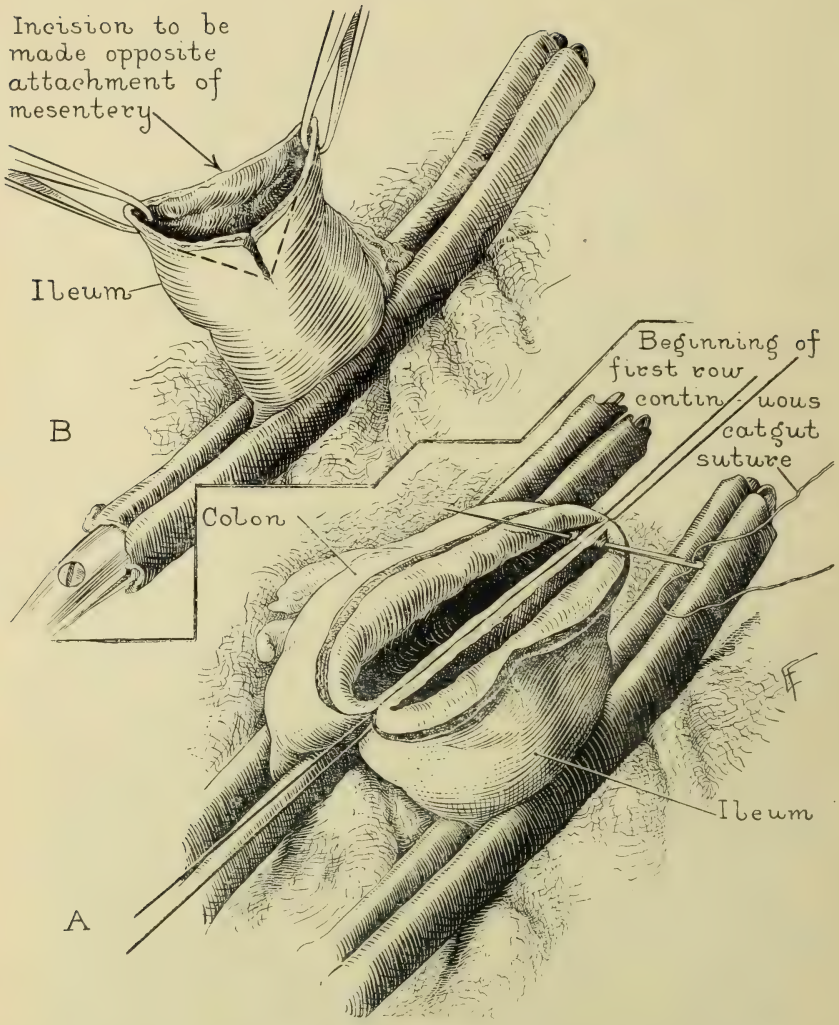

FIG. 2.—*A*, ileum and colon aligned and posterior row of sutures begun; *B*, method of increasing caliber of ileum when not of sufficient size for direct anastomosis.

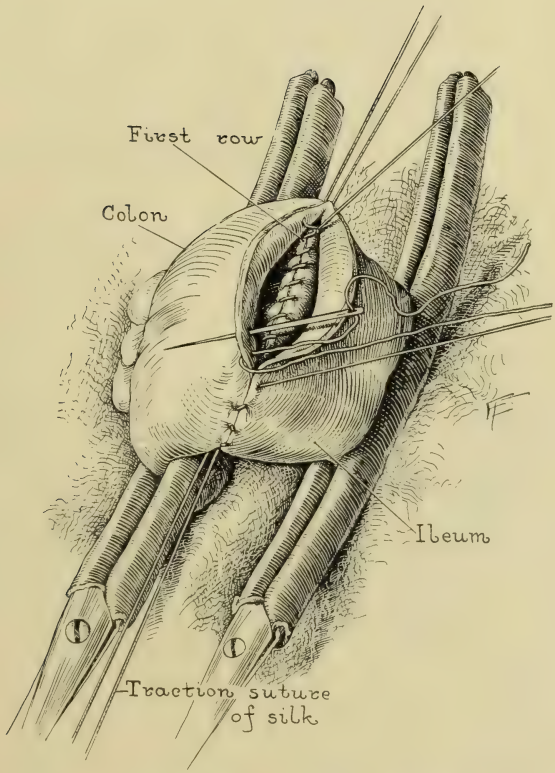

Fig. 3.—The posterior suture completed and the anterior suture begun.

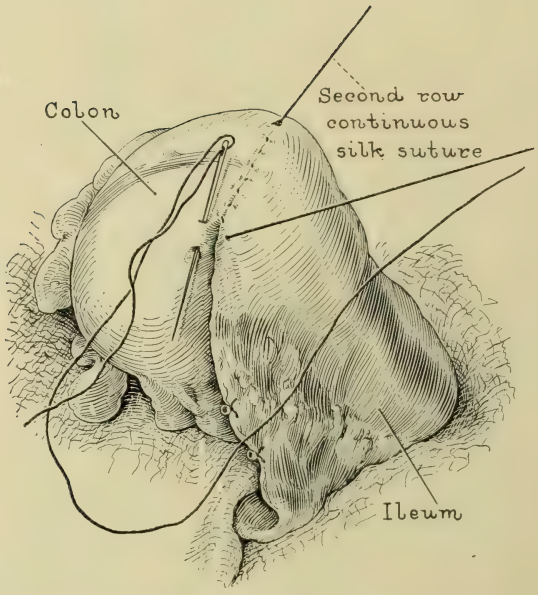

Fig. 4.—Second suture of silk completing anastomosis anteriorly.

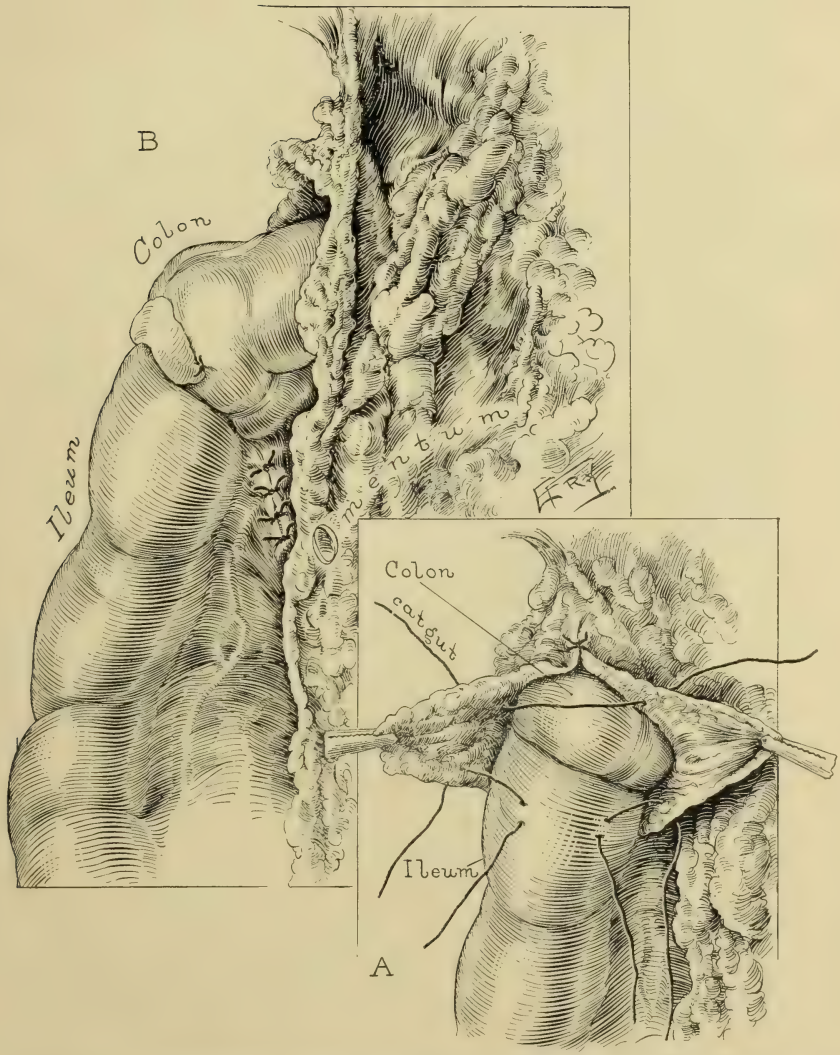

Fig. 5.—*A*, anastomosis completed and omental cuff prepared for fixation over suture line; *B*, anastomosis completed and protected by fat tag, when omentum is not available.

covered clamps are placed on each segment of bowel two or three inches from the end, the clamps at the extremity of the intestine are removed and by repeated swabbing both ends are thoroughly cleaned out. The ends of the intestine are now approximated by two traction sutures, each of which is placed about half-way between the anterior and mesenteric borders (Fig. 2, *a*). They serve a most important purpose in maintaining a correct alignment and in preventing untoward narrowing of the anastomosis during suturing. Redundant mucous membrane is trimmed away with scissors until all coats of the bowel are flush with one another. The union of the segments is then accomplished by a continuous suture of chromic catgut. This suture begins laterally from the mucous side, including all the coats of the intestine, first uniting the posterior walls (the stitching being greatly aided by traction on the sutures originally placed) and continuing anteriorly around the circumference of the bowel by the C. H. Mayo method until the starting-point is reached (Fig. 3). Two points deserve emphasis in connection with this suture: (1) the stitches should be taken close enough to the cut-edge of the bowel so that the circulation will not be impaired and (2) no catgut should be left exposed. A careful cleansing of gloves and instruments, and the anastomosis should now be made. A reinforcing musculoperitoneal suture of fine silk is placed around the anastomosis (Fig. 4), the two borders of mesentery are brought in apposition by catgut sutures and, finally, the anastomosis is protected by available omentum or fat tags (Fig. 5). This protection may be most satisfactorily made by utilizing a collar of omentum (which can be stripped back before the intestine is resected) (Fig. 1) to surround the anastomosis very much as the intestine itself is used to protect the suture-line in the tube method of anastomosis. The entire field is now cleansed, gloves and instruments are changed and the wound closed without drainage. Drainage predisposes to fistula and should be omitted unless exceptional circumstances demand it.

The results obtained by end-to-end anastomosis between ileum and colon conducted in this manner have been excellent, and, having proved the safety of the method, its obvious simplicity is sufficient for its recommendation. We believe that if the operation is carried out with strict attention to every detail the utility of axial union of small and large intestine will be quite apparent.

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HERNIA

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I have taken this occasion for a personal review of my long studies of hernia. While yet a medical student I was intimately associated with a young doctor who was a severe sufferer from congenital hydrocele. The inguinal canal was somewhat imperfectly developed. The peritoneal opening was so small that the fluid gravitated slowly into the sac and gave little trouble except after some hours of standing. Then the suffering was very considerable, relief following the recumbent position. In the morning the fluid had usually disappeared. There was no hernia. This condition is so rare that many active practitioners have not met a single case.

We consulted Dr. Henry J. Bigelow, of Boston, then New England's leading surgeon. Operation was not to be considered, but the so-called White truss was advised. This truss had a specially strong spring governing the pressure of an ivory pad in the hope that inflammatory adhesion might cause obliteration of the narrow peritoneal connection and yet not seriously injure the spermatic cord. I watched the result with daily interest. With real heroism the sufferer bore the pressure continuously day and night for a considerable period, until, indeed, the pad was half-buried in the surrounding edematous tissue.

Today we should consider an effective cure certain by a comparatively simple operation, involving no danger. Any opening into the peritoneal cavity, no matter how slight, was

then deemed too hazardous for serious contemplation. The study of this rare condition of imperfect congenital development caused me to devote my attention to the subject of hernia.

For some years an assistant of Dr. J. B. Jackson, in the Harvard Medical School, I had the exceptional opportunity for anatomical and pathological study. It soon became apparent that by far the more common cause of inguinal hernia in the male was a lack of proper congenital development of the inguinal canal. Such study of pathological conditions had its fruitage upon my return to America after extended postgraduate work, the latter portion of which was with Mr. Lister as his first American pupil. My first operative cases were for strangulated hernias soon after my return in 1870.

Mr. Lister had ligated arteries, although utterly ignorant of the work of one of the first American surgeons, Dr. Jameson, of Baltimore, who published original studies as a prize essay in 1827. Mr. Lister, dominated by the teaching of Pasteur, practically repeated the work of Dr. Jameson, arriving at similar conclusions. Dr. Jameson was a remarkably painstaking, careful surgeon. It is evident that most of his operations were conducted with special attention to cleanliness. Under the inspiration of his teaching many surgeons on both continents ligated arteries with animal sutures cut close, and buried in the wound. Septic conditions too often supervened. On this account an otherwise most noteworthy chapter in the history of surgery was neglected and forgotten. At the time of my pupilage, Mr. Lister had never used the buried animal suture, although it now seems a natural corollary to his buried absorbable ligature for occluding an artery. My first operation, using buried catgut sutures, is historic and marked an era in my own surgical experience.

In February, 1871, I closed the abdominal wound of a strangulated hernia in the approved way. The late Dr. A. P. Clark, of Cambridge, for whom I operated, said: "If you

leave the wound in this fashion, the intestines will be in the bed before morning"—(because of the coughing induced by severe bronchial asthma). I then closed with buried catgut sutures, followed by primary union, with perfect recovery which was permanent. I spent a comparatively sleepless night wondering if irreparable injury had been done, so strong was the force of previous teaching and experience.

I had equipped a laboratory with two assistants for bacteriological study for the purpose of differentiating if possible the varieties of septic infections, then known to be accompanied by various degrees of virulence and danger. Guinea-pigs and rabbits were utilized in observing the resulting physiological processes in aseptic wounds closed by sutures of various kinds. Although this is not the place for making more than a reference to the results obtained, which have been published elsewhere, the conclusion was reached that normal aseptic structures held in coaptation without undue constriction would be followed by primary union. Well-vitalized tissues in any portion of the body could thus be successfully reconstructed by aseptic absorbable animal sutures.

The histological processes occurring in the tissues thus enclosed are of exceptional interest. The suture is surrounded by leukocytes which invade the suture material and little by little are themselves transformed or replaced by connective-tissue cells, thus holding the parts in place and reinforcing them by a permanent band of living connective tissue. Such reunion is of special importance in the restoration of the inguinal canal.

The prenatal development of the inguinal canal is particularly instructive. Prior to the seventh month of intrauterine life, the testicle normally remains within the abdominal cavity. As if by change of purpose in differentiating the sexes, the testicle is slowly withdrawn through the abdominal wall and placed externally in the scrotal pocket. In normal development this change is perfected by the con-

struction of the inguinal canal in such a manner that the intra-abdominal pressure closes the walls of the canal at a right angle to its long axis. If this process is complete, inguinal hernia is of rare occurrence; indeed, I believe, except in a traumatic way, it almost never takes place.

The works of the old anatomists show that these facts were generally recognized, but I find no reference made to them as a special provision for the prevention of hernia nor to the lack of proper development as its cause. The earliest of the great writers on hernia who, in an imperfect way, recognized the anatomical deviation from the normal was Cloquet, of Paris.[1] This industrious student made five hundred dissections of the inguinal tract, with a careful tabulation of his observations, only to arrive at the important fact that in a certain percentage of the cases in which hernia did not exist there was a depression of the lower border of the inguinal ring, with a more or less pronounced bulging outward of the peritoneum. This he called the infundibular process and described it as a normal condition.

Even now it is asserted by some surgeons that this condition is normal and that it can be demonstrated in strong, healthy subjects with a firmly closed inguinal canal. In abdominal sections in men it is easy to show that the peritoneum smoothly covers the vas deferens and its vessels as they emerge from the abdominal cavity, and that the internal ring is not marked by a depression of it. When tension is made on the cord, depression is apparent, as would be expected, since the relationship of the parts necessarily admits of a certain amount of mobility. This, however, is not that which Cloquet emphasized, showing, as figured and described, a real process of the peritoneum within the lower border of the internal ring much depressed—an incomplete closure of the canal. It is still more commonly regarded as anatomically normal. A minute examination, however, will show that in all these instances the inguinal canal is foreshortened and its obliquity lessened. Hence the

intra-abdominal pressure is of necessity more or less deflected in the line of the canal instead of at right angles to it. Owing to this deflection, inguinal hernia usually occurs and, in my opinion, is its cause, although not appearing until the later periods of adult life, even in old age. When occurring very late in life the cause will be found in lessened resistance, dependent upon degenerative changes in the surrounding structures. It is important that anatomists correct the mistake of considering the infundibular process in any proper sense a normal structure.

In the upright position the weight of the abdominal contents is deflected forward by the promontory of the sacrum in a way to lessen greatly the gravity pressure upon the pelvic organs. The recti muscles form a strong supporting column with their basic anterior pubic attachment. Muscular strain, brought to bear with any degree of violence upon the abdominal wall, causes a deflection of the compression force in nearly equal degree from the promontory of the sacrum, on either side, to the aponeurotic attachment of the muscles to the pelvic bones. Thus it will be seen that when normal conditions exist this force must ever act in right lines to the inguinal canals. Were it not for this it would be difficult to understand why the normal condition of man, as an upright animal, should not be that of a hernial protrusion of the abdominal contents along the lines of a necessarily open inguinal canal.

The reproduction of the wonderful dissections which we have been considering demonstrates these facts, but perhaps we are more deeply indebted to Sir Astley Cooper than to any other for pointing out this remarkable provision of nature for the production and maintenance of this normal equilibrium of function and retaining force. He demonstrated that the transversalis fascia lying immediately beneath the peritoneum was normally so reinforced and strengthened as to form a strong posterior wall to the inguinal canal, and that it also, by the dissemination of its fibers about the cord, made

a firm boundary to the internal ring, thus producing a fixation point of definite character for the exit of the cord from the abdominal cavity.

The other most notable use of an oblique opening to produce closure of normal structures, by force applied from within the organ, is the passage of the ureter into the urinary bladder. In a less degree, because internal pressure is necessarily less, nature uses the same principle of construction in the formation of the biliary and pancreatic ducts. But for this wise provision the functions of these organs would be nugatory and hernia would be the rule rather than the exception.

Based upon and profiting by this knowledge it seems demonstrable that the only sure method of cure for inguinal hernia in man is the restoration of the inguinal canal to its normal obliquity which, by bringing the intra-abdominal pressure upon the parts, not alone deflects this force from the line of the canal but firmly closes it by pressure upon its adjacent walls. The most marked example I have ever seen of this applied force was in the case of the great athlete, Sandow, who, lying in a horizontal position, lifted by a single effort a barrel of flour attached to each foot. With him the abdominal muscles were developed to a far greater extent than in any other athlete known to me.

My own first study of this anatomical principle was in my early boyhood, when my play-fellows, engaged in football, used the inflated bladder of a bullock. The question was earnestly discussed as to why the air did not escape after tying the urethral opening, and if no air passed out, how it was possible for the urine to enter the bladder. The debate fired my determination to make an anatomical demonstration. Having surreptitiously obtained a gun, and shot a squirrel, with a knife, scissors and one of my mother's knitting needles, I gave my first anatomical lecture. The effect of this investigation was of value in my initial studies upon the inguinal canal.

The demonstration of the obliquity of the inguinal canal was clearly made by the early anatomists. The plates of the great Andreas Spigellius[2] in his masterly work clearly show this. In a sort of blind way a number of the early surgeons made efforts to restore the obliquity by operating through the canal without opening it. Perhaps the most noted and successful operator was the famous John Wood, of London. By a most ingenious technic he attempted the closure of the posterior wall of the canal by silver-wire sutures. Near the close of his life he wrote me that he had operated 339 times with only 7 deaths. This is all the more remarkable since his operations were before the aseptic regime.

In 1890 the opportunity for making a very careful examination of John Hunter's wonderfully preserved dissections was also my privilege. They are the admiration of every anatomist. The arteries, veins and lymphatics were injected with hot wax preparations and are seemingly in as good a state of preservation as 150 years ago. His preparations show correctly the normal obliquity of the inguinal canal.

The great master of all time in the study of hernia and its surgical cure was Sir Astley Cooper, preëminently the first surgeon of his generation. His great work in two folio volumes was first published in 1804 and 1827. The expense of his illustrations alone was five thousand pounds sterling. They are all life-size and all are the work of eminent artists. It had been my purpose to reproduce this magnificent work which is now so rare that only few practitioners have even seen it. I am the fortunate possessor of both editions. The publishers—Longmans & Company, of London—for whom the original copper plates were made, found, after a search, that only recently these plates had been melted up. So far as they could ascertain, I had been the only inquirer for them for seventy-five years. Fortunately I have been enabled, through the generosity of the Appletons, to reproduce a considerable number of these illustrations by the heliotype process. These reproductions were the work of the Helio-

type Company, of Boston, and cost something in excess of $6000.

Not until the establishment of aseptic surgery, thereby ensuring the safe reconstruction of many parts of the body by the use of the buried absorbable suture, was it possible to profit by the great storehouses of knowledge furnished us by the many workers in this field of anatomical research. A brilliant chapter of surgical procedures has been contributed by members of the medical profession in America. Our famous Boston surgeon, John C. Warren, was the student of Sir Astley Cooper and was probably the first American surgeon to operate for strangulated hernia. Operations for the cure of hernia had never been seriously discussed. Indeed, all operations that were in any way associated with the opening of the abdominal cavity were held in surgical reprehension. From the day of McDowell to about 1875 only a few bold pioneers had ventured to put into execution any means for its surgical cure.

The first case of ovariotomy in Cambridge that had ever recovered was my patient. More than one of the senior practitioners criticized it, saying, "What a pity that she had not died. On the encouragement drawn from the recovery, that enthusiastic young surgeon will kill a dozen women."

I established a hospital of my own for the purpose of solving these new problems. A previous mortality in abdominal sections of about 40 per cent., as established by different American operators, was reduced to 4 per cent., and nearly every operation was without suppuration. Repeated cases of successful results for the cure of hernia demonstrated the necessity of a free surgical dissection, the removal of the peritoneal sac and the reconstruction of the inguinal canal to its normal obliquity. Convinced of the danger pertaining to the use of catgut for suture material, and by no means satisfied with its anatomical structure—a thin layer of connective-tissue fibers obliquely interlaced, hampered with the difficulty of rendering it sterile—I entered

upon a series of anatomical investigations to find tendons suitable for surgical purposes. The psoas muscle of certain animals is a multiple muscle, each fascicle having its own independent tendon extending to the extremity of the tail. This is beautifully illustrated in the squirrel and in the rat.

By the courtesy of my late friend, Dr. Hunter McGuire, of Richmond, I obtained an opossum and was delighted to find a similar anatomical construction. Believing that all the marsupials would have the same type of anatomical development, my researches of several years were at last rewarded by obtaining tendons from the tail of the wallaby, a species of small kangaroo. I found that in the marsupials the anterior group of the lumbar muscles comprise a series of separate fasciculi, each fasciculus having its independent tendon extending to the very end of the tail. Each animal furnished twenty-five to thirty complete distinct tendons of about uniform size and of great strength. My first supply was obtained in 1882, since which date no other suture has been used in my own surgery. After much difficulty they were procured in quantity sufficient for general use. Few surgeons who have obtained them have returned to the use of catgut except as a necessity. The entire Australian supply is now commandeered by the English Government for military purposes.

I have operated upon patients of all ages from a few months to over eighty years, but upon few of the latter class, however, except after strangulation, where in every instance the reconstruction of the inguinal canal has been made. The operative procedures in a general way are to secure a restoration of the structures to their normal development. The peritoneal sac is opened and held on tension and closed off in the long axis of the opening by a continuous double tendon suture. Of course those structures which form the posterior wall of the canal are most important and vary considerably in their anatomical arrangement. The transversalis is sometimes found entirely surrounding the spermatic cord, making a

foramen through it. As a rule, however, it lies entirely behind the cord. The internal oblique muscles are evenly coapted and sutured from below upward. The spermatic cord is held on tension and the suturing is continued until the internal ring is reformed. In this way the peritoneal opening is elevated to its full extent. It is rather surprising how closely the structures beneath the cord may be closed without interfering with the circulation of its vessels. This part of the operation must be done with extreme care, making close approximation of the strong structures which go to make up the posterior wall of the canal.

The deep layer of the skin is rejoined with a fine tendon and a sharp Hagedorn needle. The suture must penetrate the deeper layer of the skin only, since penetration of a hair bulb or sweat duct may lead to infection. When care is used to introduce the needle exactly opposite the emergence of the previous stitch, advancing only by the length of the part penetrated, a slight tension upon the suture will render the coaptation accurate, leaving the edges of the skin in even approximation. The first and last stitch are fixed by a buried loop knot. The external wound is now covered with contractile iodoform collodion, reinforced by a few fibers of cotton. The ends of the suture are cut off and allowed to be withdrawn beneath the skin, and the point of emergence is closed by the iodoform collodion seal. This seal is antiseptic; when properly applied it renders skin infection almost impossible.

Dr. Maynard, of Dedham, Mass., in 1846, gave to the profession, as a surgical dressing, collodion—a discovery made incidental to his research work in the investigation of guncotton. The discovery has proved to be of much greater value than was at first supposed, since its use in septic wounds, then almost invariably the rule, was detrimental rather than otherwise. An aseptic wound, aseptically closed by aseptic, buried, absorbable sutures is kept aseptic by the use of the collodion seal. Iodoform, in solution, inhibits the develop-

ment of the Micrococcus pyogenes albus in the skin. The seal is loosened only by the proliferation of the epidermal cell and is absolutely trustworthy for a week or ten days. Such a seal may be reapplied, but an aseptic wound thus aseptically treated has in this period undergone processes of repair and is beyond the danger of subsequent infection.

The measures here recommended apply equally well to all aseptic wounds in all parts of the body, but they were first used and their values demonstrated in my early operative work for the cure of hernia.

Aseptic animal sutures, aseptically applied in an *aseptic* wound, *aseptically* maintained. This is the *sine qua non* without which more or less of failure must ensue. Otherwise, buried sutures are not to be commended in any wound, and must ever be a source of danger. The advantages of the above method of operation and closure of the structures for cure of hernia are obvious: First, by no other means than deep closure of the parts by the use of sutures to be left in the wound and not to be subsequently removed—*buried* sutures —can the posterior border of the inguinal canal be restored, the parts strengthened and reinforced, or even the neck of the sac closed, and the redundant tissue removed.

The way in which the suture is applied for the reinforcement of the parts and the structures to be included for this purpose vary considerably. I have felt for a long time that a double continuous suture is to be preferred, and for this reason devised the method of sewing in which the suture is applied by the use of a curved needle with eye near the point. The first stitch is taken by entering the point of the needle through the lower border of the attachment of the conjoined tendon and the inferior posterior border of Poupart's ligament. The needle is unthreaded, the suture centered and tied in a half-knot, the needle rethreaded with the opposite end of the suture and withdrawn through the opening made at its entrance. Continuous sutures, to the number of four or five, are applied in this way, until the last stitch closes firmly the inferior border of the internal ring quite upon the cord at

its exit from the abdomen. The last stitch taken, the suture is tied upon the side and the ends cut short. It must ever be remembered that the object of the suturing is to hold the structures in even approximation without tension, and the chief danger from suturing lies in an undue constriction of the tissues. When properly applied, it will be observed that the transversalis fascia has been reinforced by the attachment of the posterior border of Poupart's ligament to the posterior border of the conjoined tendon, thus forming a new strong wall of the inguinal canal.

The cure of inguinal hernia by the above-described procedure is simple, easy, and dependable. This is impossible except by a free dissection and an intelligent reformation of the structures involved. The suture must be aseptic and absorbable. By far the most satisfactory material is carefully selected tendon, that from the tail of the kangaroo being incomparably the best yet discovered. The aseptic suture must coapt well-vitalized aseptic structures, while the wound must be maintained aseptic. Primary union will follow. Thus the thousand-year-old problem of surgery has been satisfactorily solved.

If this were the proper place for its discussion, I would gladly review the experience of many other surgeons who have attempted to solve this same problem by various modifications of the method advocated. In large degree failure has followed because they have omitted to familiarize themselves with the proper coaptation of the parts involved, using only the usual variety of surgical needles. I did not myself realize the importance of the full curved needle with the eye near the point, until, some time ago, demonstrating the method in one of the large hospitals before the members of a National Surgical Society, the only available surgical needles were rather long straight ones provided by the hospital.

A bare reference to the necessity of accurate anatomical knowledge on the part of the surgeon is all that is required in this paper. I have seen the femoral vein injured in several instances. It goes without saying that no member of our pro-

fession should assume the role of the surgical expert unless he is duly trained to the discharge of his most responsible duties. This is the reason why a few of us seniors organized our new surgical society and have entitled its members to be called "Fellows" of the American College of Surgeons. Under the pressure of great demand for membership it is more than possible that the number has been extended quite beyond a wise conservatism. One of the great advantages resulting from this organization is that, in a general way, it aids materially in filling the ranks of the surgeons called to military duty and assures a reasonable preparation for the performance of the duties involved.

When to Operate. When I began operating for hernia in 1871, I was met by objections amounting to disapprobation, as during preceding generations operation was deemed unjustifiable except in strangulated cases. Even such cases were usually undertaken after a long period of waiting, undue efforts having been made for reduction, and the vitality of the incarcerated intestine either greatly impaired or destroyed. Such operations were of course followed by a high rate of mortality and operative measures for the cure of hernia were discredited.

The first efforts of cure were, by a more or less uncertain method of closing, a very considerably enlarged abdominal opening. Next followed operations for incarcerated hernia. Here, as a class, all varieties of trusses and supports were of little avail, and the sufferer was allowed to go on, becoming more and more of an invalid, for the most part incapacitated for labor. Encouraged by success with such cases, I adopted the rule that all should be operated upon. These irreducible hernias included two most noteworthy examples where the larger portion of the intestines was retained in the hernial sac which extended nearly to the knee. Both proved fatal owing to the unexpected consequence that the pressure of the returned intestines into the abdominal cavity was so great as to impair the heart's action and respiration, and death followed in a few days although the wounds were non-infected.

Then for a considerable period there followed an ever-widening conviction that all children should be wisely operated upon when the conditions were in any particular degree marked. My experience in operation upon children I consider as perhaps the most successful of all. Then came the truss-bearing army, numbered by many thousands. It is my present conviction that operation for cure is advisable for all men, otherwise healthy, who are subjects of hernia to any considerable degree. In my many publications upon hernia, little by little, I have outlined and discussed these various conditions at much greater length than many surgeons have thought necessary. There can be no doubt that future operations for the cure of hernia will be the rule. Aseptically operated upon with skill, the danger to life must be regarded as very slight. In an experience of over forty years I have never seen a fatal case except where the integrity of the intestines had become involved. The buried absorbable suture, first used as a necessary factor in the reconstruction of the obliquity of the inguinal canal, must ever be considered, when aseptically applied, as the first great factor of cure.

I have sometimes thought that the cure of hernia was my best contribution to surgery, but I am more and more impressed with the belief that the buried absorbable animal suture, in its general application to a diversity of uses, is of major importance. It is wisely adapted to be used with great profit in nearly every aseptic wound, and abdominal surgery could have in no other way reached its present remarkable perfection. My abdominal operations alone include nearly five thousand cases and in hardly a single one has the buried animal suture been omitted, as a primal factor in the control of hemorrhage and the reconstruction of the structures involved.

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INTRA-ABDOMINAL AND SLIT HERNIA, WITH OBSTRUCTION

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In this brief report of 7 cases selected because of their unusual nature there is 1 true internal hernia. The other 6 are styled slit herniæ, as they were protrusions of intestine through accidental apertures in the mesentery, omentum, gastrocolic ligament and adventitious membranes. Three of the series were postoperative and 3 postinflammatory.

Observations on internal herniæ cannot well be based upon a large series of cases, for the pages of medical literature record relatively few such reports. Treitz[1] in a most interesting monograph first called attention to internal hernia, emphasized the truth that they may occur in fossæ that are normal, and related the probable method of their development. Moynihan[2] has condensed the knowledge of the medical world on internal hernia from the time of Treitz, to whom he gives full credit, up to the present day. He describes in detail the anatomy, pathology and surgery of the various types, and the accessibility of his monograph makes further description unnecessary. To us the most important observation noted by the various authorities is that nearly all diagnoses of internal hernia are either postoperative or postmortem, and that but few have been brought to operation before intestinal obstruction has supervened.

There have been previously reported 20 cases of hernia through the foramen of Winslow, the first in 1823 by Blandin,

found at autopsy. The most recent review was made by Jeanbrau and Riche.[3] In the opinion of Moynihan one or more of four anomalies must be present. (1) A common mesentery for the whole intestine; (2) absence of the secondary fusion of the ascending colon to the posterior abdominal wall; (3) abnormally large size of the foramen of Winslow; (4) abnormal length of the mesentery, giving rise to undue mobility of the intestine.

Prolpase into the lesser peritoneal cavity may also occur after imperfect closure of the mesocolic aperture following posterior gastro-enterostomy. Protrusion through the foramen of Winslow most surely requires a more violent physical effort than any other form of internal hernia, the majority of which no doubt develop quite gradually and progress to acute obstruction less abruptly.

Case I.—*Hernia through the foramen of Winslow.*

R. S., aged twenty-one years, Potosi, Mo., glass worker. Occupation required heavy lifting in a stooping position. His health has been perfect up to December 20, 1912. While at work he was attacked by such severe abdominal pain that he was removed to his home on a stretcher. Persistent pain, constant nausea and vomiting and progressive shock continued for thirty-six hours. No cathartics were given and enemata were ineffective. His physician finally diagnosed obstruction and brought him to the hospital.

Examination. Pulse, 120; temperature, 99.6°, having suddenly dropped from 101.2°; leukocyte count, 19,000. Abdomen evenly distended, sensitive, visible peristalsis, area of dulness in right flank.

Diagnosis. Acute obstruction with peritonitis. Promptly explored, under difficult anesthesia.

Findings. Diffuse peritonitis; small intestine greatly distended. Tracing up from the cecum at a distance of about twenty-four inches, the ileum entered the foramen of Winslow, the distended proximal loop emerging at the same site. A large boggy mass could be felt within the lesser peritoneal

cavity, pressure upon which resulted in a free flow of bowel contents from the foramen along the examining finger. Attempt to reduce by careful traction proved futile, only about ten inches of the distal bowel responding.

The abdominal cavity was flushed with sterile water, the foramen drained, the proximal loop drained through a Paul tube and abdomen closed with multiple drains. The drainage from the intestine was largely hemorrhagic. Patient was under light anesthesia twenty minutes and rallied nicely for four hours after the operation. Eight hours afterward, following a profuse bloody discharge through the intestinal drain, he collapsed and died. Autopsy was refused.

In all the reported cases of hernia into the lesser cavity the symptoms have been identical. Severe epigastric pain, vomiting, shock and collapse. Temperature and pulse as in all beginning obstructions, until the integrity of the prolapsed bowel, are impaired. Perforating ulcer or gall-bladder may have the same agonizing pain, shock and collapse, but temperature rises rapidly as soon as contamination occurs.

Obstruction should be diagnosed early, but to clinically differentiate between a foramen of Winslow hernia and any of the retroperitoneal types is practically impossible.

Successful results in the cure of hernia at the foramen of Winslow are exceedingly rare. Jeanbrau and Riche describe a somewhat impractical method of enlarging the foramen to release the contents of the lesser sac. Moynihan advises that collapse of the pocketed gut be obtained by draining it through a mesocolic incision and then reducing through the foramen. In a case explored early which resists reduction at the foramen an acceptable method of procedure would be to clamp and resect the herniated loop at the foramen, tie off and remove the resected section through a mesocolic incision below the greater curvature of the stomach, drain through both openings and establish temporarily an artificial anus with the viable gut-ends, reëstablishing continuity when advisable.

The percentage of acute intra-abdominal lesions that would fail to respond favorably to well-directed surgical intervention instituted early would be exceedingly low. That degree of so-called scientific ambition that will permit a physician to stifle an unmistakable cry for help is incomprehensible in its selfishness and inimical to the best interests of the sufferer.

There are certain absolute signs of intestinal obstruction. As soon as they are demonstrable, what matters it whether it be three inches or three feet of bowel involved; whether it be an unusual type of retroperitoneal hernia or a tiny fibrous band constricting the lumen of the intestine? When our eye is invaded by a foreign body or a constriction about our neck impedes our respiration we instantly remove the offending agent, if possible, and determine its physical qualities at our leisure.

Mortality in the surgery of acute obstruction will remain high until we thoroughly realize that early diagnosis and immediate relief are the only factors that will materially lower it. The medium by which we can most surely dispel the mists of obscurity in acute abdominal lesions is the diagnostic exploratory incision.

In the following 6 cases of slit herniæ, 4 were acute and 2 chronic obstructions. Five recovered and 1, an ileomesenteric hernia with volvulus and acute obstruction, died. Peritoneal adhesions, membranous pericolitis and adventitious veil formations bear an important relation to the etiology and pathology of these cases, and a discussion of the development of such anomalies might be of value did not the necessity for brevity and the observations of Hofmeister, Connell, the Mayos, Binnie, Jackson, Hertzler, Crossen and others make it inadvisable to inadvertently open up for discussion a field that deserves more concentrated attention.

There is but little doubt that congenitalism, infection, trauma and transmigration of bacteria in intestinal stasis contribute to the formation of so-called adventitious veils,

but the author is at present more directly concerned in the timely recognition and correction of the resultant pathology.

CASE II.—*Jejunomesenteric hernia.*

Mrs. D., aged thirty-five years. Family history negative. Multipara. General health gradually declining since last confinement, eight years ago. Physical and *x*-ray tests elicited symptoms of marked intestinal stasis and chronic obstruction. Stomach was distended with pylorus fixed high, lesser curvature two fingers below the umbilicus, greater curvature within the pelvis, resting upon the transverse colon. Gastric motility low, retention and periodical emesis indicating high obstruction. (Detailed findings omitted.) As she was fast losing weight and her condition was unimproved by lavage, medication and rectal feeding, exploration was requested.

Operation. February 22, 1916. Right rectus incision; chronic appendix removed; gall-bladder normal; greatly dilated stomach in subumbilical area; pylorus high and firmly fixed by postpyloric and duodenal adhesions, but admitted two fingers when stomach was elevated; marked ptosis of both hepatic and splenic flexures of colon. There was no intestinal distention, and in the opinion of both the internist and operator the acute angulation at the pylorus occasioned by the sagging stomach and colon was the cause of the partial obstruction. It was evident that the hypotonic stomach could but feebly perform its function except when the patient was in the recumbent position.

The suspension operation of Coffey, modified to meet the conditions, attaching the transverse colon and its flexures to the parietal wall, and shortening the gastrohepatic omentum, was rapidly done. Operative recovery was uneventful. Patient was permitted to sit up in fifteen days, with firm abdominal support, placed upon a carefully selected fat-producing diet and sent home on the twenty-fourth day. Improvement was gratifyingly rapid.

In May, three months after operation, while arising from a

stooping position, something "gave away," accompanied by a sharp pain and nausea. She felt better the following day, but gradually lost what she had gained and chronic obstruction slowly returned. She suffered abdominal pain at intervals, but no local symptoms developed. During June and July she declined rapidly in vitality. Fluoroscopic examination showed stomach and colon well supported and motility greatly improved. Her condition so nearly approached acute obstruction that she was reoperated on August 8, 1916.

Operative Findings. The transverse colon was supported at points of attachment by ligamentous bands. The angulation at the pylorus was satisfactory, but postpyloric adhesions and increased thickening of the duodenum looked suspicious of ulcer, and gastrojejunostomy was decided upon. The lower loops of the ileum were somewhat distended. While exposing the jejunum for a posterior anastomosis only about eight inches could be elevated. On visualizing the field it was discovered that through a rent in the mesentery of the jejunum, ten inches from the angle of Treitz, the jejunum disappeared, twisting itself almost to occlusion at this point. Between nine and ten feet of small intestine were withdrawn after freeing the surrounding adhesions. The bowel wall was everywhere intact, showing no ill result from its imprisonment, except at the point of torsion, where an acute obstruction was evidently developing. The slit was closed and a wide postgastro-enterostomy done.

Although her operative recovery was unretarded she failed to improve as rapidly as desired, having developed an achylia. In the hands of a competent internist she is now enjoying good health, with a gain in weight of twenty pounds.

In this case the primary symptoms were chiefly due to marked visceral ptosis, intestinal stasis and high partial obstruction, caused by the sharp angulation at the pylorus and angle of Treitz. The suspending operation of Coffey gave temporary relief, the progress ceasing when the jejunal

obstruction developed. The hernia did not exist at the first operation. The jejunum was exposed but not handled, as gastro-enterostomy was not contemplated.

CASE III.—*Ileomental hernia with volvulus.*

Mrs. W., aged thirty-three years. History of chronic appendicitis, with several acute attacks, during one of which a "lump" had developed in her right side and confined her to bed for several weeks. She was brought to the hospital on a stretcher, with the classical symptoms and history of an acute obstruction of twenty-four hours' duration.

Operative Findings. Omental tumor attached to the subcecal fossa and right adnexa by fibrous adhesions of long standing. Perforating the omentum over the site of the cecum was a loop of ileum, six inches long, covered with plastic exudate, and dark in color. It was easily reduced, for there was but little pressure at the aperture, the obstruction being due to a torsion of the herniated loop. The loop was enclosed within a wet sponge and relaxed within the abdomen during the removal of the appendix and omental tumor. It reacted perfectly and the wound was closed without drainage. Recovery uneventful and no further trouble in three and a half years.

CASE IV.—*Ileomesenteric hernia with volvulus.*

Mr. C. W. D., aged forty-five years. Entered hospital in state of collapse. Patient's diagnosis of bowel block of four days' duration verified by symptoms. Was immediately explored.

Operative Findings. Diffuse peritonitis, with intestinal contents and blood free in the abdomen. Liberal adhesions in right lower quadrant, omentum not participating. A twelve-inch loop of ileum, gangrenous and collapsed, lay in the subcecal fossa. It was attached to the tip of the appendix by a fibrous band and was twisted once upon itself at the mesenteric opening through which it passed at a point about seven inches from the ileocecal valve. The adhesions were freed and the entire loop was brought out through the wound

and excised with double tube drainage. Despite free abdominal and intestinal drainage and a liberal supply of fluids intravenously the patient died on the following day.

It was evident from the nature of the adhesions binding the loop to the appendix that the prolapse through the mesenteric slit had existed for some time and that the torsion had precipitated the attack. His condition was so precarious from the time of his entry that a satisfactory history was not elicited.

CASE V.—*Ileal hernia through adventitious membrane and femoral hernia.*

Mrs. B., aged eighty years. Five years ago I operated on this patient for acute obstruction, due to kinking of small bowel in a mass of adhesions about the cecum, in the depth of which was a chronic appendix. She enjoyed good health until April 25, 1916. In consultation I found her again developing symptoms of obstruction. Because of her age expectant treatment was resorted to, but in four days obstruction was complete and patient vomited persistently. She was operated on April 29.

Operative Findings. The only adhesions remaining from the operation five years before were between the anterior surface of the cecum and a loop of ileum. An inflammatory membrane spread from the right wall over the cecum and superimposed loop and was attached below to the fundus of an atrophied uterus. The free end of the adherent loop of small bowel penetrated this veil, twisted 180° upon itself at the aperture and disappeared in a thumb-sized femoral hernia, from which it was easily withdrawn. The discoloration at the point of torsion disappeared after five minutes' relaxation; the femoral sac was inverted from within and the ring closed by suture. Patient went home on the fourteenth day and is at present in excellent health.

This is the oldest patient operated by the author for acute obstruction. She reacted from the anesthetic and necessary manipulation in a most pleasing manner.

CASE VI.—*Postoperative hernia and ileal hernia through adventitious membrane with chronic obstruction.*

W. B., aged twenty-eight years. Ten months ago patient was subjected to an operation for acute appendix. He was attended by his local practitioner, who merely stabbed and drained. A fecal fistula persisted for a time, closing and opening at intervals. Obstipation for four days preceding his operation October 3, 1916.

Operative Findings. A large-sized postoperative hernia was present, with a discharging sinus in the center. The scar was removed and the adjacent reparative tissues prepared for overlapping operation. Terminal ileum, cecum and ascending colon were enclosed in a mass of adhesions. Three distinct kinks of the ileum bound by fibrous adhesions were released. An adventitious membrane spreading from this mass down to the site of Hesselbach's triangle was pierced by a knuckle of small bowel to the depth of three inches. No adhesions confined it there. While elevating the cecum the patient coughed and vomited, and on each expulsive effort the loop would invaginate and reduce itself freely. Adhesions were freed, the appendix removed and the hernia closed. The patient is working at his trade as a blacksmith without inconvenience.

CASE VII.—*Ileo-gastric-colic hernia.*

Mrs. N. H., multipara, aged thirty-one years. History of severe attack of peritonitis following abortion several years ago, confining her to bed six weeks. Gradual deterioration in health since that time. Constipation with four- and five-day periods of obstipation. Pain and constant tenderness over the entire abdomen; mucous colitis, several attacks of arthritis and all the symptoms of severe intestinal stasis were recorded. There was an immovable mass in the pelvis. Fluoroscope depicted nothing of interest, except a sagging stomach and a sharp angulation of the transverse colon in the lower right quadrant.

Operative Findings. (February 25, 1913.) The most interesting picture of postinflammatory membrane formation I have ever observed was present in this case. A smooth, glistening, transparent membrane extended from the hepatic flexure obliquely downward to the sigmoid and on to the bladder, virtually an accessory peritoneum enclosing all the structures of the right lower quadrant. This was divided, exposing a fixed retroverted uterus and adnexa and a tumor below the cecum the size of a large orange. When this mass was elevated it was found to consist of eight inches of transverse colon which had been pulled below the level of the cecum and held there by an adherent omentum. The appendix was atrophied and clasped within the narrow thickened omental body and was crossed by several fibrous bands. A six-inch loop of ileum enclosed in a sac had insinuated itself through the gastrocolic omentum in the angle of of the V-shaped transverse colon. It was adherent at the aperture and irreducible without dissection. After releasing the omentum and colon and removing the appendix, eight sharp angulations of the terminal ileum were demonstrated within two feet of the ileocecal valve. The hernia comprised a short loop three and a half feet from the cecum. The patient went home on the eighteenth day and is at present (five years later) enjoying good health.

The particularly interesting feature of this case is the unusual variety and distribution of postinflammatory sheaths gliding smoothly over each other, adherent to structures only at either end. One other feature that is often seen, but still mystifies, is the number of acute ileal angulations all densely adherent with only a mild state of constipation resulting. Thus is supported the contention that circulatory interference is a prime factor in acute obstruction.

At the operating table one is often caused to marvel at the ability of nature to surmount mechanical obstacles. Knowledge of her leniency in this regard should make us eager to respond to her signals of distress when overwhelmed. More

important by far than the cause of death in obstruction is the necessity of locating and removing the cause itself, even though to accomplish this purpose we find it necessary to acquaint the public with the danger signs of all acute diseases of high mortality. Both cases that ended fatally in this report, could have been saved by early interference.

How discouraging it would be to mortality statistics if surgical interference, when indicated, could be instituted at the right time. Wonderful progress in the prevention and cure of tuberculosis and allied diseases has been made through persistent educational efforts. We should cast aside our squeamish fear of public criticism, circumvent the quack and the public press and extend this crusade to include accurate and intelligible information on all diseases, acute and chronic, the neglect of which is dangerous, and while applying the education to the public, reënlist ourselves as students under the banner of timely surgery.

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A NEW OPERATION FOR COLON STASIS MODIFIED FROM ROVSING'S, WITH A REPORT OF TWENTY-EIGHT OPERATED CASES

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In many of the cases of stasis the colon resembles closely the sharply angulated condition of a rubber tube looped and suspended from two nails placed some distance apart.

The stasis is in inverse proportion to the height of the fixed points and the length of the loops.

The mesentery of the transverse colon is more or less fan-shaped and envelops the longitudinal band on the posterior surface and extends downward almost to the band found on the lower border of the colon. The third band is anterior and just below the attachment of the omentum. When we have a long so-called redundant colon these bands are very much elongated by stretching.

The only step in this operation which exactly duplicates Rovsing is the three rows of sutures put in the anterior surface of the stomach for elevation and fixation of the stomach, and our claims for a new operation are based on (1) the preservation of the omentum intact, (2) the actual suture of the colonic bands to the stomach, (3) the lowering of the flexures and (4) the elevation of the liver by pulling the suspensory ligament up through the fascia and suturing it as if done in the Gilliam operation.

Operation. The first thing to be done is to examine the cecum, and if much dilated, narrow by suturing two bands

together up to the hepatic flexure. The appendix should be removed.

The stomach is then brought up and three rows of strong linen sutures are put in across the anterior surface, care being taken not to go too deep and to avoid wounding any bloodvessels. When the x-rays show a very high attachment of the flexures, and especially the splenic, this should be pulled down and incised and dissected down far enough to prevent any angulation after the colon is raised. A point is selected on the ascending colon which will bring it up snugly to the lower border of the stomach near the pylorus; a similar point is selected on the descending colon and sutured to the stomach a few inches below the cardia. The band on the lower border of the colon is found and a midpoint is selected and the omentum is held up and by transmitted light this point on the colon is sutured to the stomach through the omentum midway between the two sutures mentioned above (heavy linen is used); a midpoint is again selected on the two halves of the colon and stomach and points are again selected between these sutures until all of the slack is taken up in the colon (which contracts down so that it fits snugly to the lower border of the stomach), and until the sutures are within at least half an inch of each other.

As the mesentery extends down almost to this band we find it narrows the colon somewhat by suturing it to the stomach and forms an apron which holds the colon up in place and gives a smooth surface beneath the mesentery. The omentum has not been injured in the least and falls down in its normal place and prevents any adhesions.

The three stomach sutures which were left long are now threaded on a Reverdin needle and brought out through the abdominal wall near the border of the ribs on the left side and a little nearer the midline and at a lower level on the other side. These sutures are drawn up tightly and clamped close to the skin and the abdomen is then closed, and the sutures are then tied over a tin box about three inches square

wrapped in gauze (I usually get one of the boxes in which the Natural leaf tobacco comes).

The patients are kept in bed four weeks and the sutures removed in from three to four weeks.

I have operated on 28 cases by this method with 21 cures, 2 improved, 1 not heard from. Four cases died, none of them caused by the operation. One case died of pneumonia (grippe) two weeks after operation; 1 case died of acute dilatation of the heart one week after operation; 1 case died four weeks after operation, probably from an acute exacerbation of pellagra, and the other case of inanition two weeks after operation. Two cases have been operated four years, 4 cases three years, 13 cases two years, 3 cases one year, 2 cases six months; 26 cases in women, 2 cases in men.

In 8 cases the ligament operation was done on the liver and the left lobe of the liver was sutured to the diaphragm in 1 case. The cecum was pleated in 8 cases. Two of the cases were four months pregnant and went on to term with a normal delivery. In 1 case a gastro-enterostomy was done.

THE NEW WAY

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The activities of the first year of our country's participation in the great war have been recorded. What will be history's final estimate of our efforts and accomplishments? Will it contain a chapter on the rudderless effort of a great nation to accomplish a task without a plan and by commonplace methods, or will it record an era in history when a new way was evolved by the guiding hand of a great leader to meet the exigencies of an unusual demand? Are we now too much interested in the details of this struggle to estimate the fundamental methods and activities employed in its execution? Are we too near the scene of action to gain a proper perspective of the field? Are we too much committed and trained in the routine of old statesmanship and politics to be willing to admit that a new statesmanship has successfully replaced the old? May we not presume to compare the contentions of the ultraconservative mind with public opinion and thus estimate the methods and tendency of progress?

The conservatives have been loud in their condemnation of non-partisan, volunteer workers, as they have been employed by the Government in meeting its emergency. They have questioned the advisability of the Government's recognition and support of the frank methods of handling vice among civilians and in the Army. They have questioned

the practicability of the future treatment of the problems between nations by our great leader.

Congress acted more wisely than it knew when, in August, 1916, it created, at the request of the President, the Council of National Defense and the Advisory Commission, the latter to serve without compensation. The appointments to the Commission of the seven civilians were made by the President and, lo! there was about an equal representation of the two dominating political parties, if one is able to judge at all of the politics of these men by their attitude in action.

While the Council and the Commission were being created as a peace-time machinery, war clouds were hovering on the horizon. After three meetings of the Council and the Commission the nation was involved in the greatest war of history. Every department and bureau of the Government, directly or indirectly engaged in the conduct of the war, was in the midst of the greatest activity and expansion of its existence.

Clearly the task for which the Council of National Defense was created was at hand. Its duties had already been forecast. Every department and bureau welcomed assistance. What was more natural than that the Commission should immediately suggest that the best experts in all lines of endeavor should be brought to Washington to fill in the gaps in the expansion work of the Government? It was apparent that not only the work of actual warfare would have to be performed by a civilian army, but that the enormous work in the departments at Washington would have to be supplemented by men who had never before been engaged in Government service. Therefore, the Commission, each one of the seven members acting as an expert, said: "We will call in men as we have been called; we will call in men of marked ability and experience who will serve disinterestedly and without compensation." This program was adopted. Thousands of men, upon request, came to Washington to confer, to advise and to take off their coats and work from ten to fifteen hours a day. Many of them have not only worked without

compensation, but have paid their own travelling expenses and upkeep in Washington. In expanding the many bureaus in which they have been particularly interested they have called to their assistance members of their own home organizations, their managers, assistants, stenographers and clerks and have turned them over to the Government. The last question asked by a competent worker is: "What compensation will I receive?" and the last question asked about him is, "What are his politics?"

And thus this new way has developed in a great emergency, and the Government is being served by disinterested experts from all parts of the country. Each of the seven civilians, with only advisory authority and without compensation, succeeded in nine months in bringing to the city of Washington first a hundred men of influence who would not accept salaries, but were willing to serve the Government patriotically without compensation. This number was increased many fold until seven thousand such men were serving in Washington. The advice and services of these men aided in accomplishing the transition from peace-time conditions to the present war-time expansion, until now the parks and waste places of Washington have been occupied by temporary buildings, accommodating thousands of workers, the overflow from the reorganized bureaus. It is difficult to realize what has been accomplished by this great army of patriotic workers; to realize the magnitude of the task of enrolling two million men into the military department of the Government; to realize the difficulties involved in expanding purchasing departments, supplying provisions, clothing and equipment for these two million men; to appreciate what has been accomplished in spite of all criticism in providing explosives, ordnance and fighting material for an Army and Navy of two million men. If you will summarize these facts in your own mind and estimate the extent of this accomplishment you will then appreciate that much of the criticism of the Government for executing this enormous task in so short a

time in the only possible way was due to the fact that critics could not conceive that so vast and great an undertaking could be executed by men without partisan fear, without official positions, without salaries, and in many cases without hope of receiving credit for the work done. Many minds cannot comprehend such a sacrifice, nor will they admit that it represents a new and a better way.

THE NEW WAY OF COMBATING THE VENEREAL DISEASES. The Government's program for the protection of the soldiers against vice and its attendant diseases has been the result of a rapid evolution from the old method which merely acquiesced in what was termed the inevitable to a new method of combating an existing evil that already gives promise of far-reaching and beneficial results.

The first step was a sympathetic attitude on the part of our administrators toward the enforcement of a law against the use of alcohol in the Navy, when Secretary Daniels issued his famous order on that subject. Clear-headed athletes seldom go wrong morally if they are awake to the consequences and if their brains are unaddled by alcohol. The Daniels order, with its proof of wisdom, followed by the wholesome legislation making it a misdemeanor for any fighting man to imbibe alcohol, maintains the brain in a normal state, and the teaching of the Fosdick Commission in enlightening the men as to the consequences.

This movement has the support of the administration, of Congress and of all right-thinking people, and a marvellous organization has been established to deal with the whole subject of protecting not only our fighting forces, but the cities and rural communities as well, from the results of an evil which has proved itself more life-destroying than the ravages of actual warfare.

It is strange that the most insidious opposition attempted to block this new way. All traditions of centuries were entrenched against it. "Personal liberty" was again to be violated by the ruthless hand of the reformer. Respectable

complacency was to be aroused from its sleep of conventionality. Men were to be made milksops and a single standard of morals was to be enforced for all. But the arguments of science, clean living and a great war to which fathers and mothers were to lend their sons, aroused public opinion until it was decided to "give the new plan a tryout," and the tryout has been so dramatically successful that a great spiritual light has dawned and has illuminated the dark places and driven the greatest evil of civilization to bay. The new way has not produced a milksop and, on the other hand, the rounder in war and society has been eliminated. In his place has come the clear-eyed, strong-limbed, self-respecting athlete with a knowledge of himself and a high sense of moral and spiritual obligation to his fellow-men and women.

The successful prosecution of this new way by the American Government will save the lives of more men and women who would die of loathsome diseases in the next ten years than have been destroyed by German guns during the past four years. The actual trial of the system in this one year of war has conclusively proved this fact.

Nations at War. And now comes the *new way* of dealing with nations at war with one another which threatens to smash the traditions of centuries and to establish the principle which was so difficult for the followers of the Great Teacher to understand twenty centuries ago. The strong nations are to yield to the weak. "Jockeying for balance of power" is to yield to the establishment of a balance of power that will compel strong nations to deal equitably with the smaller ones.

Early in February, President Wilson made the following statement: "Our whole strength will be put into this war of emancipation—emancipation from the threat and attempted mastery of selfish groups of autocratic rulers. We believe that our own desire for a new international order under which reason and justice and the common interests of man-

kind shall prevail is the desire of enlightened men everywhere. Without that new order the world will be without peace and human life will lack tolerable conditions of existence and development. Having set our hand to the task of achieving it, we shall not turn back."

These remarks were supplemented by him in a recent speech in Baltimore as follows: ". . . Germany has once more said that force, and force alone, shall decide whether justice and peace shall reign in the affairs of men, whether right as America conceives it or dominion as she conceives it shall determine the destinies of mankind. There is therefore but one response possible from us: Force, force to the utmost, force without stint or limit, the righteous and triumphant force which shall make right the law of the world and cast every selfish dominion down in the dust."

Thus says our chief, who is now one of the recognized spokesmen for civilization and who has created the spirit that is behind this new way—this new way which, when spoken of by him, seems the only way.

Work of the Advisory Commission of the Council of National Defense. I am not here to speak of the general work of the Government, of the Advisory Commission or of the Council of National Defense, but of that particular work which interests us as medical men. When the war began the Medical Department of the Army consisted of between four and five hundred officers and the Medical Department of the Navy of about the same number. There was a law in existence making it possible for the Surgeon-General of the Army to enroll civilian doctors as Medical Reserve Officers of the United States Army. The Medical Section of the Council of National Defense was organized to aid in the enormous task of expansion of the bureaus of the three Surgeons-General and to suggest ways and means for caring for the health of men in civil and industrial life. A plan for a General Medical Board was adopted, upon which were placed representatives of the

civilian medical profession, the Surgeons-General of the Army, the Navy, the Public Health Service and representatives of the Red Cross; officers of the principal societies—surgical and medical—of the country. This consisted of the Presidents, Boards of Directors or Boards of Regents of the American Medical Association, the American College of Surgeons, the Clinical Congress, the American Surgical Association, the Southern Surgical Association and other like bodies. The representatives on this Board were requested to act by the Secretary of War upon my recommendation. The active membership of this important board was depleted from time to time by men being placed on active duty, many of whom are now serving in Europe. This board holds a session once a month, and through a well-developed system of committees, almost everything of importance in the conduct of war, from the stand-point of medicine and sanitation, is discussed and recommendations are presented. These recommendations are, in turn, presented to an Executive Committee on the following day, this Committee consisting of the Surgeon-General of the Army, the Navy and the Public Health Service, the Chairman and Vice-chairman of the General Medical Board, Dr. Victor C. Vaughan, Dr. William J. Mayo, Dr. William H. Welch and Rear Admiral Cary T. Grayson. The chairmen of the various committees make recommendations. If these recommendations are approved by the Executive Committee they are taken to the Council, and if approved by that body are distributed in the way of information to those in authority in the bureaus concerned.

State and County Organizations. When the Advisory Commission was appointed the Medical Section took over the organization of the Committee of American Physicians for Medical Preparedness, consisting of committees of medical men in each State. These committees have been amplified until in each State, as you know, a strong committee of physicians, known as the State Committee, Medical

Section, Council of National Defense, is prepared to cooperate in every way with the central body. These committees are appointed by the Government and are a necessary clearing machinery between the home doctors and bureaus and other activities of the Government. As long as the war lasts and the services of doctors are required these committees will represent us.

In each county of the United States has been organized a County Committee, which places us in touch with all County Medical Societies. Through this rather complicated, but very effective, organization we have been able to place much to the credit of the Advisory Commission. Our accomplishments may be summarized as follows:

We have succeeded in enrolling 21,000 civilian doctors for the Medical Reserve Corps.

We have aided through the Munitions Board and other agencies connected with the Advisory Commission in obtaining supplies for the Medical Departments of the Army, the Navy, the Public Health Service and the American Red Cross.

Through conferences with experts, called at different times, which conferences appointed committees and coöperated with the Surgeon-General, we have prepared and published standard tables of instruments, drugs, hospital supplies and hospital equipment. These tables carefully record by number all materials made in this country that are available for our work.

One of the conferences of Deans of Medical Schools succeeded in adjusting the difficulties in enrolling a large number of civilian doctors as reserve officers from among the teaching forces of the medical school.

Another conference discussed in detail the proper handling of the venereal diseases and the relation of the alcohol problem to venereal diseases. This conference resulted, indirectly, in the appointment of the Fosdick Commission and of our strong Committee in the Council of National

Defense dealing with the venereal disease problem and emphasized in the Surgeon-General's Office the importance of this problem.

Volunteer Medical Service Corps. Two matters of unusual importance have been taken up by the General Medical Board of the Council of National Defense recently. One is the organization of a Volunteer Medical Service Corps.

Briefly, this consists of men who have offered their services to the Surgeon-General, and who, for physical reasons, have been rejected, and for medical men over the enrollment age who are willing to do some kind of service for their Government, preferably along military lines. The original recommendation of the Committee formulating the plans for this service stated that this organization could be of service to the Government in the following ways:

1. By aiding in the work of selective enlistment. This is now being done by the Medical Advisory Boards, many of whose members should be eligible to the Volunteer Medical Service Corps.

2. By aiding in general as consultants wherever consultation may be necessary for enlisted men.

3. By maintaining in the best possible condition the medical services of hospitals, medical colleges and laboratories depleted by absence of those in active duty.

4. By reclamation of registrants rejected for physical unfitness under the Selective Service Law.

5. By caring, so far as possible, for the families and dependents of enlisted men.

6. By aiding in the general sanitation of the country.

The formal recommendations for the organization of the Volunteer Medical Service Corps, accepted and approved by the Council, are contained in the following resolutions:

Whereas, There are many physicians registered in the United States who are ineligible for membership in the Medical Reserve Corps on account of physical disability, overage, essential public or institutional needs; and

WHEREAS, In view of the need for the services of these physicians in civil and military places it is considered desirable by the medical profession of the country and the General Medical Board of the Council of National Defense, including the Surgeons-General of the Army, Navy and Public Health Service, that some use be made of the services as offered; therefore be it

Resolved, That the Council of National Defense authorize the Medical Section of the Council of National Defense to organize a Volunteer Medical Service Corps to include in its membership those physicians eligible for membership in the Medical Reserve Corps on account of physical disability, overage, essential public or institutional needs and women physicians for the purpose of making available the services of such physicians in any way deemed advisable by the Surgeons-General of the Army, the Navy and the Public Health Service or the Council of National Defense; and be it further

Resolved, That an appropriate insignia be authorized to be worn by the members of the Volunteer Medical Service Corps, the form of such insignia to be designed by the Medical Section of the Council of National Defense.

The important organization, in all its details, will be under the direction and authority of the General Medical Board of the Council of National Defense.

COMMITTEE ON INDUSTRIAL MEDICINE AND SURGERY. The second innovation to which I refer is the establishment or authorization by the Council of a Committee on Industrial Medicine and Surgery. The necessity for a committee of this sort is to coördinate the agencies authorized to carry out the provisions as follows:

1. To provide against unnecessary human waste in industry and society during the war.

2. To offset the drain on industry of man power caused by raising of military forces.

3. To meet the need for greatly increased production.

4. To avoid preventable deaths and disabilities from accident and disease.

5. To restore to full producing power in the shortest possible time sick and injured workers.

6. To increase output by maintaining workers in good condition.

7. To provide healthful places in which to work.

8. To provide healthful homes and communities in which to live.

9. To meet shortage of medical service induced by military needs.

The resolution asking for the creation of such a committee states definitely the activities to be represented. It reads as follows:

Be it Resolved, That there be created an Advisory Committee on Industrial Hygiene, which shall be made up of a representative from the Public Health Service, acting as Chairman, a representative from the following Federal Agencies:

Department of Agriculture.
Department of Interior.
Department of Commerce.
Department of Labor.

And a representative from each of the following:

Organized Industry.
Organized Labor.
Organized Medicine.
Organized Industrial Medicine.

Responsibility of the Civilian Doctors. This brings me to the consideration of my last problem. It is not generally realized or appreciated that 29 out of every 30 medical officers among the fighting forces of our Army will be civilian doctors. This places an enormous responsibility upon the civilian doctors. In order to carry out adequately these responsibilities it will be necessary for the reserve officer to have rank that will place him somewhere near the same

footing as the regular medical officer. After the civilian doctor has been in the service for one year, there certainly will be no disparaging his services in any place he may be asked to occupy as compared to the regular medical officer. At that time, if the reserve officer is ever to become a soldier, he will be in a position to fulfil his entire responsibilities. If he is to do the work side by side and shoulder to shoulder with the regular officer for the good of the service he should have equal rank. When we take into consideration the sacrifices of the average civilian doctor who leaves his business, leaves his practice and serves his country for a year or two in a conflict of this kind to come back after the war with his business dissipated and with new responsibilities to assume, we have one more strong argument in asking that the reserve officer should have full rank as soon as he is entitled to it with the regular army officer. Fortunately, our chiefs, the Surgeons-General of the Army and the Navy, are definitely with us in our desire for equal rank. Surgeon-General Gorgas has urged repeatedly since the beginning of the war that a law be enacted that will enable him to give proper rank to the strong men who have volunteered their service in his department, and he must have thousands of civilian doctors serving under him if he has a properly organized Medical Department.

The following letter shows how the Commander-in-Chief of the Army and Navy, the President of the United States, stands on this important subject:

"My dear Dr. Martin:

"I read very carefully your memorandum of February 27 about the rank accorded members of the Medical Corps of the Army and have taken pleasure in writing letters to the chairman of the Military Committees of the House and Senate, expressing the hope that the bill and resolution may be passed.

"Cordially and sincerely yours,

"Woodrow Wilson."

Two bills exactly alike were presented in Congress on February 5—Senate Bill No. 3748, introduced into the Senate by Senator Owen, and House Resolution No. 9563, introduced into the House by Mr. Dyer; these bills are similar to the one introduced by Senator Owen last year as an amendment to another military measure. Please note that the present bill reads as follows:

That hereafter the commissioned officers of the Medical Corps and of the Medical Reserve Corps of the United States Army on active duty shall be distributed in the several grades in the same ratios heretofore established by law in the Medical Corps of the United States Navy

The Surgeon-General shall have authority to designate as "consultants" officers of either corps and relieve them as the interest of the service may require.

That means, interpreted into more understandable language, that there will be a general officer, either a Major-General or a Brigadier-General, for each 200 medical officers, or 5 general medical officers to each 1000 medical officers; or, for our present enrollment of 15,000 medical officers, 75 general officers and the usual proportion of Colonels, Lieutenant-Colonels, Majors, Captains and Lieutenants that are provided now for the Regular Army.

In closing may I be pardoned for making a direct reference to the personality of our two chiefs, representing the Army and the Navy—namely, Major-General Gorgas, Surgeon-General of the United States Army, and Admiral Braisted, Surgeon-General of the United States Navy. Could anything have been more fortunate for the medical profession than to have this war begin with two such men on the job—each of whom had already an international reputation as scientists a medical man and an indefatigable worker?

You may depend upon it when criticisms come, as they have come, and as they will come in the future, that these two men will be the first to make every effort to correct mistakes that are avoidable and to make it impossible for them

to be repeated. Nothing so cleared the atmosphere as the prompt action by Admiral Braisted when there came trumped-up criticisms of a hospital ship, in ordering immediately an investigation by the strongest civilian sanitarian. The shortcomings were admitted, and, with equal frankness, it was stated that they would not occur again. So, likewise, the recent report of Surgeon-General Gorgas when his work was criticized in which he definitely stated the fact, indicated the causes, and located the responsibility.

Carry-on. This is the time for the earnest prayerful searching of one's soul. It is the time for prayer that precedes action. Each hour of every day of every week there is something that one can do that will help to stop the Hun. Create in your mind the consciousness of shame that will make you hesitate to eat white bread; to waste the necessities of life; to use automobiles and able-bodied drivers for pleasure riding; that will compel you to carry your own hand-bag; to stop unnecessary travel; to conserve your fuel; to, above all things else, think every minute how you may play the game of "carry-on" by your sacrifice and example.

After you have done all this and all else that you can think of; after you have given yourself in service; after you have given your son to the fight; your daughter to the Red Cross; your best efforts in thought and action to conserve your Government's and your Allies' resources—then you must give of your wealth if you have it and of your small means if you have not wealth. The rainy day has arrived. The collection is now on. Your Government that has protected you—your home is in peril. If you have given, give again, give now.

Finally, may I appeal to the medical men of this city and the medical men of this country who are capable of doing their bit either in the service of the Army or Navy, or in the work of caring of the medical schools, the hospitals and for the civilian population, to enroll in the Medical Reserve Corps of the Army or Navy or in the Volunteer Medical

Service Corps, which is now open to those who have been rejected for physical reasons or who are above the military age. Let me appeal to you to be enrolled and in uniform or be ready to wear the uniform upon call or to accept the insignia indicating that you are in the Volunteer Medical Service Corps, and that you have a good reason for not wearing the uniform or are not ready to wear the uniform. In making this appeal I wish to emphasize the importance of this new Volunteer Medical Service Corps.

Please do not criticize the heads of the Administration until you know the facts. Visualize in your mind the enormous task that has been accomplished by the Government since April 6, 1917. Appreciate that this great work could not have been done without a few mistakes in details. Don't criticize our Secretary of War, who has been back of the great organization plan and who has helped to develop the new way of doing things—viz., employing the services of thousands of non-political volunteers in the organization of the Service, in their great task of expansion from peace time conditions to war time necessities. Realize that in war time many of the most important accomplishments are not published—they are secrets of war. Instead of criticizing, write letters of commendation to overworked and trusty officers who have accomplished so much, in order to renew their courage and add to their strength. Thank them for their steadfastness of purpose and for their optimism based on the consciousness of good work done. Thank them for working fifteen hours a day in their great efforts to prepare for battle. When you prepare to criticize—stop—search your libraries and read the comments upon Mr. Lincoln and Mr. Stanton published in the partisan press from 1861 to 1865.

SUBPARIETAL RUPTURE OF THE INTESTINES FROM TRAUMATISMS TO REDUCIBLE INGUINAL HERNIÆ

By Randolph Winslow, M.D., F.A.C.S.
Baltimore, Maryland

Subparietal rupture of the intestines is a comparatively rare injury. The small intestine is much more frequently ruptured than the large, and the ileum is the usual seat of the laceration, though the jejunum and duodenum are also injured in a progressively diminished ratio. The causes of the rupture may be either direct blows on the abdomen, whereby the bowel is compressed against the bony structures, or a force which causes bursting of the distended viscera by a sudden displacement of their gaseous or liquid contents. While direct violence is the common cause of the injury, it is known that indirect violence may also cause this lesion, such as a fall upon the feet or the buttocks. Emanuel Senn reports an interesting example of the latter injury in the case of a woman who while walking fell to the ground, striking her right buttock. At operation a small opening was found in the jejunum. The actual causative traumatisms are numerous, such as blows from narrow blunt objects, kicks on the abdomen, falls and injuries to herniæ. While the intestine may be ruptured or even completely torn across by such an object as a cart wheel passing over the abdomen, it is more usual to find that the injury is due to a comparatively small body striking the abdomen with great force.

Gage and Lund both mention blocks of wood thrown off by circular saws as among the most common causes in New England. Kicks on the abdomen by a horse or by a man are also frequent causes of rupture of the bowel, or injuries from wagon poles or other narrow bodies. Falls on the abdomen are important causative agencies and would be likely to cause bursting of the intestine from sudden displacement of its contents. An interesting example of rupture of the alimentary tract by a fall occurred in the practice of my colleague, Dr. John D. Blake, in the case of a man who, after a hearty meal, was thrown from his buggy and ruptured his stomach, with the escape of its contents into the peritoneal cavity. Laparotomy was promptly done and the man made a good recovery.

Symptoms. The symptoms of a subparietal rupture are very similar to those of a penetrating wound or a perforating ulcer of the stomach or intestines. There is a history of a blow or other traumatism on the abdomen, followed usually by severe pain, rigidity of the abdominal walls, tenderness on pressure, vomiting, increased frequency of the pulse, with soon an elevation of temperature and a high leukocyte count. There may be marked tympanites and obliteration of the liver dulness, but these signs are by no means constant, nor is shock a constant or a reliable symptom. The patient, however, has received a mortal injury, and he generally shows it in his facies. Of course the symptoms will depend somewhat upon the extent of the injury, a small opening may give exit to intestinal contents slowly and the inevitable peritonitis may not develop as rapidly as when the laceration is more extensive.

A remarkable illustration of this fact has recently come under my observation in the service of Dr. Page Edmunds. A man fell thirty feet, striking on his back and fracturing some of his lumbar vertebræ. His abdomen became enormously distended and tympanitic. On opening his abdomen there was an escape of free gas, but no aperture could be

found in the intestines nor was any peritonitis present. There must have been a slow escape of gas from the intestines, with subsequent closure of the opening. The operation was done four days after the fall.

As a subparietal laceration of the small intestine is always fatal, so far as we know, when unoperated on, it is of the utmost importance that a diagnosis should be made at the earliest possible moment. Contusions of the abdominal wall may present somewhat similar symptoms, but they are not usually so pronounced and tend to subside, while those of a rupture become progressively more accentuated. In any case of doubt it will be safer to open the abdomen and ascertain the extent of the lesions than to wait in the hope that no serious injury has occurred.

While the general subject of subparietal injuries of the intestines is of great interest, my attention has been especially directed to the role of a hernia in the production of intestinal lacerations, since two such cases have come under my care in the last two years.

This experience was novel to me, but one would naturally think that herniæ, especially large irreducible ones, would be very liable to injury from external force, and upon looking up the literature I find a number of such cases reported. The nature of the traumatism is practically the same as has been mentioned earlier. Of 3 cases recorded by Lund,[1] 1 was due to the kick of a horse, 1 to the kick of a man, and 1 to being struck by an iron bar on the pad of a truss. H. B. Stone[2] also records the case of a man, aged fifty-one years, who stumbled and struck the pad of his truss.

In my cases one was injured by falling on the pad of his truss and the other was struck on the right side of his abdomen by an automobile. Of the 6 cases here mentioned 3 resulted from blows on improperly applied trusses. A truss that is applied to an unreduced hernia is therefore an added

[1] Boston Medical and Surgical Journal, cliii, 603.

[2] Annals of Surgery, lii, 358.

source of danger to the wearer, since it prevents the free movement of the intestine and increases the liability of rupture. While the intestine may be lacerated by direct compression between the vulnerating object on the outside and the pelvic bones behind, it is probable that most ruptures are due to a bursting from violent displacement of the intestinal contents.

In this connection Lund says: "In order to allow these ruptures by bursting to take place, both arms of a loop must be caught and compressed, otherwise the gas will not be pent in. Such conditions are obviously present in case of hernia, the loops being grasped in the rings so that the gas cannot escape, and the intestine having no muscular covering, simply the skin and the fascia of the hernia, being exposed to direct compression between the injuring body on the outside and the pelvic bones or aponeurosis, from which it cannot slip away, behind. It is obvious that intestine shut up in a hernia may be ruptured by a slighter blow than that free in the abdominal cavity."

Bunge, of Königsberg, quoted by Campbell,[1] also says, "when the contents are present in a hernial sac the free movements of the bowels may be interfered with, allowing a less active force an opportunity for inflicting damage."

It is also evident that an irreducible hernia will present conditions more favorable for rupture of the intestine than one that is reducible, and that the lesion may be less serious, as the escape of intestinal contents may be confined to the hernial sac, while similar injuries of a reducible hernia are practically certain to be followed by the escape of fecal matter into the abdominal cavity and the production of general peritonitis.

In fact, when the gut is ruptured from a blow on a reducible hernia the intestine slips back into the abdominal cavity and the hernial sac is found to be empty.

[1] Annals of Surgery, xlii, 605.

I desire to place on record 2 cases in which blows upon reducible inguinal herniæ were followed by rupture of the intestines.

Case I.—*Rupture of Intestine from Striking Pad of Truss.*

M. J., farmer, aged fifty-five years, was admitted to University Hospital on August 9, 1915, complaining of "pain in the stomach." This trouble began on the night of August 7, while the patient was running after some horses in a field, when he stumbled, fell and struck the pad of a truss that he was wearing. A very short while after this he was seized with violent cramp-like pains in the abdomen, which were only controlled with large doses of morphin. During that night and the next day he suffered with pain, some nausea and vomiting. This man lived in lower Virginia and the accident occurred on Saturday night; and as no boats were running on Sunday, he was unable to get to Baltimore until thirty-six hours had elapsed. When he reached the hospital he was desperately ill, with great pain, board-like abdomen, tenderness on pressure and other symptoms of an acute peritonitis. There was no tympanites nor was there any ecchymosis or bruising of the abdominal walls. His expression was anxious and his features pinched. His mouth temperature was 100° and his rectal temperature was 103°; pulse, 100; leukocyte count, 20,000. A diagnosis of ruptured intestine was made and immediate operation advised. A right rectus incision was made from the umbilicus downward. Upon opening the peritoneal cavity a great quantity of yellowish-brown fluid and pus escaped. The peritoneum and intestines were acutely inflamed and a widespread peritonitis was found. Floating loose in the abdominal cavity was found a watermelon seed. The appendix was found to be slightly congested, but not materially diseased. The intestines were covered with a plastic exudate and a small opening in the small intestine was found, about eight inches from the cecum. A large quantity of fecal fluid and pus was extravasated into the abdominal

cavity. The opening into the bowel was about one-half inch in length, and owing to the fibrinous exudate it was not thought that union would occur. The laceration, however, was closed with two layers of sutures, the cavity flushed out with salt solution and drainage established by means of rubber tubes and cigarette drains and the abdominal wound closed up to the drains. This patient had a somewhat stormy convalescence, but he recovered and left the hospital on September 9, one month after admission.

CASE II.—*Right Inguinal Hernia with Ruptured Intestine from a Blow.*

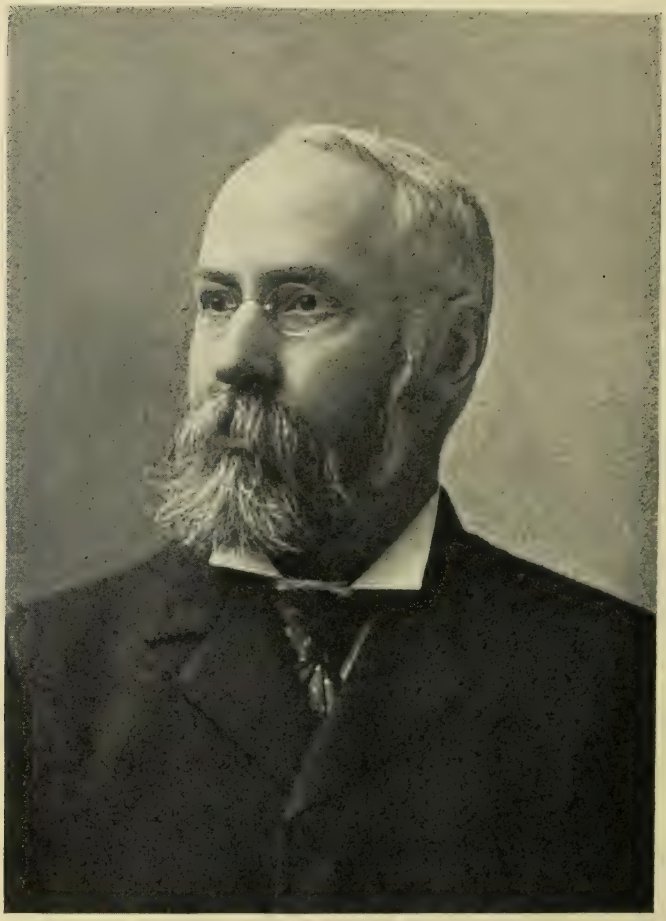
T. F., white, aged sixty years, entered University Hospital on May 13, 1917, and was discharged well on June 23, 1917.

He has been a remarkably healthy man, but has had a reducible inguinal hernia on the right side for sixteen years, which, however, gave him no trouble.

June 12, at 8 P.M., he was struck on the right side of the abdomen by an automobile. He says the hernia was down at the time of the injury and that after the injury he was unable to reduce it, though it had been easily reducible before. Immediately after the accident he had severe pains in the abdomen, which were general in character, and two hours later he became nauseated and vomited. This was repeated several times during the night and the pain became more intense. His bowels moved freely in the morning. As he had an evident lump in his right groin, his physician judged he had a strangulated hernia and sent him into the hospital for an operation.

On admission his temperature was 99° by mouth; pulse, 88; respiration, 26. There was a lump in the right inguinal region about the size of a duck egg, which was very tense and could not be reduced. The abdomen was neither rigid nor tender. The diagnosis of a strangulated hernia seemed justified and an operation was undertaken about twenty-four hours after injury. An incision about five inches in length, over the inguinal canal, revealed a large, tense,

edematous sac, very red and inflamed. This was isolated and opened, and was found to contain no intestine but a large amount of fluid and a small piece of greenish substance which resembled pickle. The walls on the sac were very thick and intensely inflamed. On looking into the abdomen a large amount of creamy pus was discovered. An incision was then made upward at right angles to the original incision, toward the umbilicus, and the appendicular region was explored. The appendix was thickened and chronically inflamed, but not ruptured, nor did it seem to be the cause of the peritonitis; it was, however, removed. The intestines were red and inflamed and bathed in pus, with patches of fibrin on their surface, but no perforation was discovered in the part examined. The stomach was also examined, but no perforation was found. I was at a loss to account for the peritonitis, but in the pus, however, were small particles suggestive of intestinal contents. The abdomen was drained with rubber tubes and cigarette drains and the wound closed up to the drains. The patient was placed in the Fowler position and proctoclysis instituted. Subsequently there was a discharge of fecal matter from the wound, quite profuse at first and gradually diminishing until it ceased and he was discharged well on the forty-second day.



LOUIS McLANE TIFFANY, M.D.

IN MEMORIAM

LOUIS McLANE TIFFANY, M.D.

1844–1917

At his country home, in Accomac County, Virginia, there passed away in October, 1917, one of the most distinguished Fellows of this Association. Elected to fellowship in 1891, elevated to the Presidency in 1895 and placed upon the honorary list in 1907, this eminent surgeon, Louis McLane Tiffany, was for many years closely identified with the work of this Association and truly represented its spirit.

He was born in Baltimore in 1844 and in that city practised continuously for over thirty-five years. After his early preparation for college he went to England and entered Cambridge University, where in 1866 he took the degree of Bachelor of Arts. Returning to Baltimore he entered the medical school of the University of Maryland and received the degree of Doctor of Medicine from that institution in 1868.

Dr. Tiffany's career as a surgeon began with his appointment in 1869 as demonstrator of anatomy at the University of Maryland. This position he held for five years, having been appointed professor of operative surgery in 1874 to succeed the late Dr. Alan P. Smith. Six years later Dr. Tiffany was given the professorship of surgery at the University and held this chair until 1892, when he resigned, becoming professor emeritus. For fifteen years he was chief

surgeon of the Baltimore & Ohio Railroad, relinquishing this position in 1903, when he retired from practice.

Besides the services to his alma mater, Dr. Tiffany held many hospital appointments. He was the first resident physician at Bay View, and he had been consulting surgeon to Johns Hopkins Hospital, St. Joseph's and the Church Home and Infirmary. He was connected with other Baltimore hospitals, and was often called upon to perform operations in other cities and States. He was a former president of the American Surgical Association, the Southern Surgical Association, the Medical and Chirurgical Faculty of Maryland and the Baltimore Medical Association. He was also an honorary member of many professional societies in this country and in Europe.

As an operating surgeon Dr. Tiffany's reputation was world-wide. One of his colleagues has said of him: "He acquired national and international fame. South of Philadelphia for years he was probably the dominant figure in american surgery, and in Baltimore and in Maryland for more than a decade he was by common consent *facile princeps*. His work was distinguished not only by its skill but by its sanity; his judgment was superb. In his study of disease he never lost sight of the individual. His touch was sure and strong and exquisitely gentle, and his authority unmistakable. He inspired confidence as few men can. Boorishness and dirtiness and manipulative roughness he despised. In a way his measure was shown 'by the grasp of his mind and by the simplicity of his means;' with a clear apprehension of the material points he was never enamored with the unessential."

It is unnecessary to review the contribution of our Fellow to the cause of surgery or to enumerate the long list of operations performed by him. He gained a high place in American surgery as teacher, author and operator. His work was of the heroic type, for nothing seemed too difficult for him to undertake, provided there was firm chance of good results.

In 1878 he removed a large nasopharyngeal polypus, which procedure is called by B. B. Browne, the medical writer, as at that time "the most difficult and heroic operation recorded in the annals of surgery."

Dr. Tiffany was built in a big mold. In his younger days he was a noted athlete, at a time when students gave little attention to athletics. He won honors in running at Cambridge and a beautiful trophy bowl was evidence of his prowess. A friend recently spoke of him as follows: "He loved his work, which he pursued untiringly and with little remission. When forced by ill-health to relinquish it he surrendered without condition and devoted the rest of his life in the same fine spirit to what he called play, always cheerful and serene and interested in everything about him through a long period with many occasions of bodily pain and distress. He greatly prized strength, but was sympathetic and indulgent in his relations with human weakness —a close and amused observer of life's foibles and fantasies. He loved Nature, of which he saw the beneficence and glory. He loved living things—the friends of birds and animals —and above all, with Goethe, he loved God and little children."

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